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EDITORIAL

DANGERS OF ROENTGENOSCOPY AND METHODS OF PROTECTION*

EVERY physician has read or heard about the many pioneer specialists in roentgenology who, after the loss of one finger after another, finally lost their lives from the spread of carcinoma which had developed as a result of antecedent injury to the skin of the fingers caused by exposure to roentgen rays repeated daily or at frequent intervals over a long period of time. It has become a habit of newspaper editors to feature each case and to glorify each successive victim as a "martyr to science." The one fortunate thing about such cases is that their number is steadily decreasing, and the reason is not far to seek. When these pioneers sustained their initial injuries, little was known about the action of roentgen rays on human tissues, and still less was known about effective means of preventing such injuries. In fact, several years elapsed after Roentgen's epoch-making discovery before the relationship between certain effects on the skin and exposure to the new rays was realized. But gradually, successive reports of similar deleterious effects caused the medical profession to understand that roentgen rays were far from harmless.

Since then, many experiments on animals and prolonged clinical observation have yielded extensive information about the changes that are induced in the tissues by irradiation under different conditions. Even laymen now know something about the danger of overexposure to the roentgen rays. Moreover, those physicians who have devoted their lives to the study of these rays or to their use for diagnosis or treatment have learned how to protect themselves and how to prevent the kinds of injury which had maimed or killed so many of their predecessors. Now that effective methods of protection are known and understood, no longer need any physician suffer injury from this cause. So true is this that,

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at the present time, any physician, and especially any roentgenologist, who suffers injury from this cause convicts himself of ignorance or of unpardonable negligence, or the burden of responsibility must rest on the hospital or other institution that forces him to work under unsafe conditions. The point is that, so far as our knowledge of the means of prevention is concerned, there can no longer be any excuse for this kind of injury.

However, to this day, the number of physicians who incur injury from overexposure to roentgen rays is much greater than is realized by the profession at large. An interesting point is that, of the total number of such injuries, the percentage among professional roentgenologists is very small indeed. The inescapable conclusion is that physicians without special training and experience in roentgenology sustain injury because they are not aware of the danger to which they are continually subjecting themselves and because they do not adopt the methods of protection without which the use of roentgen rays is fraught with hazard.

Since the war, an increasing number of internists, surgeons and general practitioners have equipped their offices with roentgenologic apparatus and have used it for the diagnosis of intrathoracic or gastrointestinal lesions, for the diagnosis and reduction of fractures, for the localization and removal of foreign bodies, or for the recognition of tumors of bone. In some instances, the physician has taken the trouble to acquire a certain amount of training, but too often his training has been confined to the instructions furnished by the manufacturer's agent who came to install the apparatus. Some physicians employ a technician to make roentgenograms, but roentgenoscopy, the reduction of fractures, and the removal of foreign bodies cannot be left to a technician, however good his training. These procedures the physician himself must conduct, if only because the weight of legal responsibility bears more directly on him. It is precisely these procedures which involve the greatest danger.

When internists watch an experienced roentgenologist examine the stomach and duodenum of a large number of patients in an amazingly short time, it probably looks as easy as golf when played by Bobby Jones. No doubt many internists go back home asking themselves why they should refer cases to a specialist when they could themselves conduct such a simple diagnostic procedure. The only cure for such naive thinking is to give them a set of clubs and let them try to beat "old man par." However, in the case of roentgen rays, "old man par" is a suffering human being and sometimes the physician himself. This also applies to the reduction of fractures and to the removal of foreign bodies. Even in the hands of a well trained and experienced surgeon, and even when a foreign body has been accurately localized and its position charted on a cross-section diagram, its removal may prove to be an extremely difficult and time-consuming undertaking. This is just as true of roentgenoscopic procedures, the apparent simplicity of which is often deceptive. As a result, thousands of patients are daily being subjected to unnecessarily prolonged exposure to roentgen rays and, incidentally, hundreds of physicians are daily exposing themselves to the roentgen rays for an undue length of time, and often with little or no protection. True, the manufacturers of roentgenologic apparatus have incorporated a measure of protection but, in the case of roentgenoscopy, few physicians know how much effective protection actually shields them from a catastrophe which may not become apparent until months or years later. If the reader thinks this is an exaggeration, let him ask any of the physicians who have been unwitting victims of this sort of injury.

If one scans the medical writings one finds that this relatively important problem is seldom mentioned. The few articles on the subject of protection have usually been written from a physical standpoint

and are too technical for the average medical reader. Many bulky tomes on the diagnosis and treatment of fractures and encyclopedias of surgery have been published by eminent physicians, not one of whom has deigned to refer to this phase of medical practice. Only one reason can explain such surprising neglect; this is that physicians in general, including all but a few of the best known specialists, have not even been aware of the existence of this problem. The unfortunate victims are not inclined to boast of the calamity which has come upon them and which sometimes has blighted a brilliant career.

The manufacturers of roentgenologic apparatus cannot escape a share of responsibility. When has any manufacturer or his agents taken the trouble to warn physicians of the danger of overexposure? Rather, the tendency has been to minimize the danger or to avoid mentioning it for fear of losing a sale. However, since those who manufacture apparatus have not known more about the subject than the physicians themselves, their share of responsibility is small. To be fair, it should be mentioned that some manufacturers have incorporated valuable protective features, such as additional lead glass besides that which overlies the roentgenoscopic screen, automatic limitation of the fluorescent image by mechanical connection of the shutter to the screen holder, complete enclosure of the underside of the roentgenoscopic table, provision for the attachment of a rubber apron impregnated with lead (which moves with the screen and constantly separates the operator from the patient), and metal cones to confine the beam of roentgen rays and to reduce scattered radiation.

For several years the general problem of protection for the patient as well as for the physician has received much attention. In the larger countries of Europe and America, the national societies of radiology have named special committees to study the different phases of the problem and to formulate recommendations. Moreover, all these societies have had representatives on the International Committee appointed to gather and disseminate information on protection. Since the first International Congress of Radiology, which was held in 1925, much progress in this direction has been made. Fairly comprehensive recommendations have been formulated and have been officially adopted. The advisability of speed in roentgenoscopic examinations, minimal palpation with the unprotected hand, the use of protective gloves, the minimal thickness of lead glass for roentgenoscopes have all been stressed, but nowhere can be found data that would enable one to know the limits of safety for the physician or for the patient.

It is astonishing that the writings of roentgenologists and orthopedic surgeons during the past ten years contain so little that might serve to warn the medical profession in general against the possible dangers of roentgenoscopy as used in reducing fractures and in localizing and removing foreign bodies.

The amount of radiation that a human being may safely receive over a long period has been estimated by several writers, but these estimates have not been based on scientifically accumulated data. The value of such estimates has been thrown into question by the fact that physicians practicing roentgenoscopy on a large scale, such as those at the The Mayo Clinic, have greatly exceeded the so-called tolerance dose for many years without any sign of injury from roentgen rays. It is obvious, therefore, that those who have attempted to establish the limits of safe exposure have been extremely cautious, and these limits probably can be accepted as entirely safe. But in view of the paucity of our knowledge, a thorough investigation of the problem seemed desirable for the following reasons: (1) to determine the time required to produce erythema of the operator's hands under the conditions that obtain when a fracture is reduced with the aid of the roentgenoscope; (2) to ascertain the degree of protection necessary for the operator's hands; (3) to find, from the standpoint of protection, the most effective combination of kilovoltage, milliamperage,

and filtration for the reduction of fractures; (4) to determine the protective value of various materials used in the construction of tops for roentgenoscopic tables; and (5) to suggest ways of preventing the serious injuries to the skin which have become all too common.

The problem has been studied chiefly from the point of view of the reduction of fractures, because this seemed most important. Many of the principles that apply to the reduction of fractures also apply to the localization and removal of foreign bodies, but in relation to fractures the position of the operator's hands differs from their position when dealing with foreign bodies.

Under the technical conditions of roentgenoscopy (voltage, milliamperage, filtration, and so forth) the majority of roentgenologists and dermatologists agree that exposure to 375 roentgens (measured in air) is sufficient to cause a mild erythema of the skin, at least in cases in which the patients are blond subjects. Since it was impossible to make the necessary physical measurements during the actual reduction of individual fractures, a standardized technique, such as that used under average conditions, was adopted. The technical conditions were as follows: potential, 80 kilovolts; intensity of current, 6 milliamperes; distance between the target of the roentgen tube and the top of the table, 16 inches (40.6 cm.); the table top, which was made of pine veneer, was ¼ inch (0.63 cm.) thick. In order to avoid ruining ordinary roentgenoscopic tubes by the prolonged operation necessary for such a study, these technical factors were reproduced by means of a mechanically rectified roentgen therapy apparatus, with an air-cooled Coolidge, broad-focus, therapy tube. A potential of 80 kilovolts represents a fair average of the voltage employed in clinical procedures. As for current intensity, 6 milliamperes exceeds the current flowing from the tube under average conditions.*

THE QUANTITY OF ROENTGEN RAYS REACHING THE OPERATOR'S FINGERS

When one grasps an extremity to reduce a fracture, with the patient in a horizontal position, one's fingers are beneath the extremity, while the thumb is on top or at the side of the limb. Therefore, the ionization chamber used to measure the rays was placed near the top of the table, where the fingers of the operator would be while reducing a fracture. The intensity of the rays was measured by means of a Victoreen roentgenometer, without any filtration, with an aluminum filter 1 mm. thick, and with a similar filter 2 mm. thick. The center of the ionization chamber, which corresponded to the center of the beam of roentgen rays, was situated 1 inch (2.5 cm.) above the top of the table. Under these conditions, and with rays filtered only by the table top, an exposure corresponding to the number of roentgens required to induce cutaneous erythema required seven minutes and forty-five seconds. When the rays were filtered through 1 mm. of aluminum, the exposure time for 375 roentgens (erythema time) was fifteen minutes and thirty-seven seconds, and when the rays were filtered through 2 mm. of aluminum, the exposure time for 375 roentgens (erythema time) was twenty-four minutes. It must always be remembered that, with all the other factors constant, the intensity of radiation is directly proportional to the number of milliamperes, inversely proportional to the square of the distance between the target of the tube and the fingers, directly proportional to the square of the kilovoltage, and decreases according to the degree of filtration. If these points are borne in mind, even if a different technique is employed, it is a simple matter to estimate the erythema dose for any particular set of technical factors.

* The large number of measurements made in the course of this study are deliberately omitted so as to avoid confusing the average physician, to whom long tables of figures are a nightmare. Those who might be interested may find these figures in another, more detailed article in the *American Journal of Roentgenology and Radium Therapy*.

THE QUANTITY OF ROENTGEN RAYS REACHING THE INDEX FINGER

In most instances of injury from the reduction of fractures under roentgenoscopic guidance, the index finger is usually injured more severely than the other fingers. This is probably because the index finger lies nearest to the central and strongest portion of the beam of rays. Actual measurement showed that the quantity of rays reaching the fingers diminished as the hand was moved away from the center of the field of irradiation.

HOW THE TOP OF THE TABLE GOVERNS THE QUANTITY OF ROENTGEN RAYS REACHING THE FINGERS

Samples of the material used for the top of fluoroscopic tables were obtained from three well known manufacturers of roentgenoscopic equipment. The top used by one manufacturer was ¼ of an inch (0.63 cm.) thick; it was composed of three-layer plywood which was 3/16 of an inch (0.45 cm.) thick; this was covered with bakelite which was 1/16 of an inch (0.15 cm.) thick. Another top, which was ⅜ of an inch (0.88 cm.) thick, was made of a five-layer plywood which was 5/16 of an inch (0.75 cm.) thick and a layer of bakelite which was 1/16 of an inch (0.15 cm.) thick. The former was used in vertical roentgenoscopic tables, and the thicker material was used in other roentgenoscopic tables.

The top employed by a second manufacturer was made of an upper layer of bakelite which was 3/64 of an inch (0.038 cm.) thick, three-layer plywood which had a total thickness of ¼ of an inch (0.64 cm.), and a sheet of aluminum which was 0.0005 of an inch (0.00125 cm.) thick and which, it is claimed, tends to disperse static electricity and filter out the soft rays.

The top of a well known fracture-roentgenoscopic table is ⅞ of an inch (2.24 cm.) thick and is made of five-layer plywood, the bottom and top sides of which are covered with bakelite which is ⅛ of an inch (0.32 cm.) thick. This table top is advertised as allowing the unobstructed passage of roentgen rays, both for roentgenography and roentgenoscopy.

From actual measurements, it is apparent that the top of roentgenoscopic tables has a definite but slight protective value. But if an equivalent thickness of aluminum is added as a filter, the slight protection provided by the table top may be discounted. However, when a table top is not used, as with some applications of the Hawley-Scanlan table, the danger of injury is slightly increased because the hands of the operator can come closer to the roentgen tube without any intervening protective element.

The filtration or protective value of each table top was measured with the same ionization chamber at a uniform distance of 16 inches (40.6 cm.), the chamber being 2 or 3 mm. above the top of the table and in line with the center of the beam of roentgen rays. Although the exposure time equivalent to an erythema dose varied slightly with each table top, the thickness of the material rather than its character was the main factor in determining the protective value of each table top. The few minutes difference in erythema time between a thin table top and a thick one may seem of small consequence but, when one realizes that doses of roentgen rays are cumulative, this difference of a few minutes, repeated frequently over years of roentgenoscopic work, becomes tremendously significant, especially since the rays that pass through the thin table top are softer and, therefore, are absorbed in greater proportion by the skin. Even a thin layer of aluminum affords valuable protection. When, as sometimes occurs when a Hawley-Scanlan table is used, no material substance intervenes between the roentgen tube and the extremity under examination, the danger of overexposure is definitely greater than it is when the tube and extremity are separated by as little as two layers of ordinary pine plywood.

THE EFFECT OF SECONDARY RAYS

Secondary radiation from an extremity during roentgenoscopy does not play an important part in the total dose. On theoretical grounds, the part of the dose made up of secondary rays should not be large, because the voltage responsible for the secondary radiation is low and the volume of irradiated tissue is small. Usually, the proportion of secondary rays is not large, and these weak, secondary rays are absorbed by the skin of the fingers or of the hand as a whole. The weakness of the secondary rays is shown further by the fact that roentgen dermatitis of the palm of the operator's hand is rare. Nevertheless, the field of exposure should be kept as small as possible.

THE DOSE OF ROENTGEN RAYS REACHING THE OPERATOR'S THUMB

In order to eliminate the possibility of a difference in absorption between a wax model and human tissue, a series of absorptive measurements were made on the forearm of an adult cadaver, which was placed on the table top, with the ionization chamber 1 or 2 mm. above the skin. These measurements clearly showed that the tissues of a patient's forearm or other part of an extremity afford a large measure of protection in comparison with the dose reaching the fingers on the inferior surface of an extremity.

AN ATTEMPT TO SPECIFY AN IDEAL TECHNIQUE FOR ROENTGENOSCOPY

The next step was to find a combination of kilovoltage and filtration which, while affording good visibility of a fracture, would at the same time afford the operator more effective protection from excessive exposure than do the techniques commonly used. With a table top made of plywood which was ¼ inch (0.64 cm.) thick, with a distance of 16 inches (40.6 cm.) between the target of the roentgen tube and the top of the table, and with a current of 6 milliamperes, variations of potential between 50 and 100 kilovolts, and variations of aluminum filters between 0.5 mm. and 2 mm. were tried, and the exposure time equivalent to an erythema dose was measured for each variation. These measurements show that an aluminum filter provides a large measure of protection, especially when the rays are generated at a low potential (kilovoltage). The value of a suitable filter is so obvious that easily accessible filters should be incorporated in all roentgenoscopic apparatus. Many roentgenologists say that, owing to the different conditions encountered during roentgenoscopic examinations, it should be possible to vary the quality (penetration) of the rays by changing the voltage at will. This can readily be done by modifying the electric factors, but the intelligent use of filtration affords more effective protection. In some roentgenoscopes the manufacturer has incorporated a filter, but usually this is so inaccessible that an attempt to change it is seldom made. However, possibility of changing the filter at will would often be a decided advantage. To this, it may be objected that an increase in erythema time obtained by increasing the filtration might lead to carelessness and the filter might sometimes be overlooked but, if the operator is made to realize that carelessness in this respect may later result in cancer, he is not likely to forget the importance of the filter.

In order to test the effect of variations in kilovoltage and filtration on roentgenoscopic visibility, five members of the roentgenologic staff of the clinic were invited into a room in which was a horizontal roentgenoscopic table of standard type. Twenty minutes in total darkness were allowed to permit their eyes to adapt themselves thoroughly. The fractured forearm of a cadaver was first examined roentgenoscopically by one of the writers, under technical factors unknown to the roentgenologists. The latter were then asked to write their opinion of the visibility and definition of the bony structure as brought out by various combinations of kilovoltage

and filter. Throughout these tests the distance between the target of the roentgen tube and the top of the table was kept at 16 inches (40.6 cm.), and the current flowing through the tube was constantly kept at 3 milliamperes. Each of the roentgenologists kept his opinion secret during the tests. When these had been completed, the respective opinions and comments of the five roentgenologists were tabulated. Unfiltered rays generated at a potential of 80 kilovolts were thought to give too bright a field. Rays of the same quality, but filtered through 2 mm. of aluminum, were generally thought to have the same roentgenoscopic characteristics as the former. Rays generated at a potential of 70 kilovolts and filtered through 1 mm. of aluminum were also thought to yield too bright a field. Rays generated at a potential of 50 kilovolts and filtered through different thicknesses of aluminum provoked a variety of comments, but all five roentgenologists agreed that rays of this quality would provide for satisfactory visualization of a fracture of the arm or leg.

From the foregoing tests, rays generated at a potential of about 60 kilovolts, filtered through 2 mm. of aluminum, and with a current flow of 3 milliamperes, should constitute the best technique for the reduction of a fracture of the forearm or leg under roentgenoscopic guidance, if both the visibility of the fracture and the protection of the operator are the controlling factors.

When the fingers of the operator were placed on the top of the table the exposure equivalent to an erythema dose, with rays generated at a potential of 60 kilovolts, filtered through 2 mm. of aluminum, and with 3 milliamperes of current flowing through the tube, was ninety-five minutes. The average potential employed by several other roentgenologists was about 80 kilovolts, and the average filtration was through 0.25 to 0.5 mm. of aluminum. The number of milliamperes varied with the part of the body examined, but not with the voltage. With rays generated at 80 kilovolts, filtered through 0.5 mm. of aluminum, and with 3 milliamperes of current passing through the tube, the exposure time equivalent to an erythema dose (375 roentgens measured in air) was twenty-two minutes. Therefore, an "ideal" technique of 60 kilovolts, 2 mm. of aluminum as a filter, 3 milliamperes of current, a distance of 16 inches (40.6 cm.), and a table top made of pine veneer which was ¼ inch (0.64 cm.) thick affords the operator's hands more than four times the protection afforded by the technique usually employed, even when his hands are in the direct path of the rays, and yet this "ideal" technique gives a better view of the fracture.

Throughout this study the emphasis has been, not on attempting to present something new, but to investigate the problem from the standpoint of effective protection for physicians who are not specializing in roentgenology, but who have to reduce fractures or remove foreign bodies under roentgenoscopic guidance. Many interns, resident physicians and general practitioners who are frequently called on to employ roentgenoscopic procedures are not aware of the danger of overexposure to themselves as well as to their patients. To repeat, the technical factors employed in the course of this investigation represent what is believed to be a fair average. These factors may be used to calculate the doses received with other techniques, but only when the conditions of exposure are identical.

CONCLUSIONS AND RECOMMENDATIONS

1. Too much emphasis cannot be laid on the danger, to the physician and to his patients, of reducing fractures under roentgenoscopic guidance.

2. The careless roentgenoscopist may, while reducing a single fracture, subject his hand or fingers to severe injury which may not become apparent until weeks or months later. He may thus expose himself to a dose of roentgen rays which, though less than an erythema dose, may later by its cumulative

effect result in injury to the skin. At the same time, the patient also may receive an excessive dose which may be masked by pain caused by the fracture and which may not be detected because of inflammation associated with the fracture, especially when the limb is in a cast.

3. From the standpoint of protection this investigation emphasizes the importance of the following points: speed of examination, minimal action of the roentgen tube, the significance of keeping the fingers away from the center of the beam of rays and taking advantage of the protection afforded by the thickness of the extremity under examination; all these factors make for increased safety in roentgenoscopic procedures.

4. The "ideal" technique employed in this study may be used as a basis for calculating the doses received with different techniques.

5. As much as possible, the hands of the operator should be kept out of the path of the rays while the tube is in action, especially when lead rubber gloves are not worn.

6. Unless the operator is careful to measure the dose of roentgen rays received by himself and by the patient, unless the work is closely supervised by an experienced roentgenologist, or unless all recommended protective measures are employed, the reduction of fractures or the removal of foreign bodies under roentgenoscopic guidance is strongly condemned.

7. Roentgen rays generated at a potential of 60 kilovolts and filtered through 2 mm. of aluminum, with 3 milliamperes of current flowing through the tube, a distance of 16 inches (40.6 cm.) between the target of the roentgen tube and the top of the table, and a table with a top made of pine veneer that is ¼ inch (0.64 cm.) thick, may not be an ideal technique but, with modifications dictated by clinical indications, seems worthy of trial.

8. Roentgenoscopic apparatus should have accessible filters which can be readily changed.

9. As suggested by Hawley, the mechanical manipulation of fractures, instead of manual reduction, deserves serious consideration.

10. Since the effects of roentgen irradiation are cumulative and signs of injury may be slow to appear, each operator should keep a permanent record of the doses to which his hands have been exposed.

11. As an additional factor of safety, each operator should calculate his maximal possible exposure and regard such a total dose as actually received, even though additional protective factors may have been employed.

12. The committees on roentgen ray protection should base their recommendations on clinical as well as on physical grounds.

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MAMMOGRAPHIC RECOGNITION OF INTRACYSTIC PAPILLOMA OF BREAST*

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THE recognition of papillomatous tumors of the milk ducts presents a tantalizing clinical problem. Unfortunately the variability of symptoms and the paucity of identifying physical signs accentuate the diagnostic difficulties. Even though Nature may sound a warning by producing a serosanguinous discharge from the nipple, our tests of precision, such as transillumination, soft tissue roentgenograms, aspiration biopsies and discriminatory palpation, may fail to locate the offending neoplasms. Any procedure which aids in determining the location, size and characteristics of these papilliferous growths is certainly to be desired.

An appreciation of the morphological characteristics of these papillomatous neoplasms affords a rational explanation of the varied clinical syndromes which they produce. A true papilloma is a localized overgrowth of the epithelial cells which line the lactiferous ducts and acini. The proliferating epithelium grows more rapidly than its connective tissue substratum and invaginates into the ductal lumen. As the tumor grows it produces a pressure dilatation of the confining duct, thus forming a localized cystic cavity. Occasionally a papilloma does arise from the wall of a preformed cyst but the great majority originate in the ampullic segments of the larger milk ducts. They grow very slowly and are usually solitary. Multiple papillomata are, however, not uncommon for they occur much more frequently than is usually appreciated. Serial sections of the breast[1] and contrast mammograms[3] have adequately demonstrated this point. When multiple, these neoplasms may arise in different segments of the same ducts or in other lobules of the same or opposite breast.

The recognition of papillomatous neoplasms is difficult as they do not produce pathognomonic symptoms. The most characteristic complaint is that of a bloody discharge from the nipple but this occurs in so many other pathologic conditions that it has but little diagnostic value unless confirmed by other evidence. A hemorrhagic or serosanguinous secretion may be associated with a malignancy,[7] papilloma of the ducts, Paget's disease, trauma, fibroadenoma, tuberculosis, chronic cystic mastitis[6] and senile parenchymal hypertrophy. To complicate matters further, a papilloma may bleed and yet the blood will be confined to the milk duct for the tumor may completely obstruct the ductal outlet. Deaver and McFarland[2] found that 50 per cent of papillomata produced a bloody discharge, 25 per cent a clear serous fluid, and the remainder did not form any type of secretion. It is apparent that the nature of the discharge has practically no diagnostic value in the recognition of duct papillomata.[7]

PAIN

Mastalgia is seldom associated with a papilloma for these tumors grow so slowly that but slight intraductal pressure is exerted. Occasionally a sudden, sharp pain may mark the onset of an intracystic hemorrhage. Multiple papillomata are more prone to cause discomfort than a single one, but pain seldom causes the patient to seek relief, and when present it has no diagnostic value.

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PALPABLE TUMORS

Unfortunately the very nature of these neoplasms renders their detection difficult. They are usually small, compressible, and frequently cannot be differentiated from the surrounding matrix. Occasionally they attain sufficient size that they may be palpated. In such instances, the tumor is usually situated in the ampullic segment of the ducts with its long axis radiating from the nipple. Pressure may elicit a point of tenderness or express secretions from the nipple. Even when the diseased lobule has been identified by the escaping discharge one can seldom feel the offending tumor. Transillumination will occasionally outline a large papilloma particularly if it is surrounded by a blood clot, but generally the tumor possesses the same translucency as the surrounding breast structures and differentiation is very difficult. Soft tissue roentgenograms, likewise, fail to isolate the neoplasm.

The failure of these papillomata to produce identifying syndromes combined with their occasional tendency to undergo malignant transformation certainly creates an interesting problem. In order to facilitate the recognition and location of these tantalizing neoplasms, a new method of roentgenographic visualization has been developed.[3,4] By this procedure, termed mammography, an accurate anatomical pattern of the ductal system can be obtained by introducing a suitable contrast medium. Any pathological condition which alters the size, shape or conformation of the injected ducts can be readily detected by subsequent stereoscopic roentgenograms.

Papillomata can be readily visualized by these mammographic studies. The stoma of the offending duct, which can be identified by the escaping serosanguineous discharge, is cannulized and injected with thorotrast and immediate stereoscopic roentgenograms are made. The entire duct and all of its branches are graphically outlined. Since these papilliferous growths protrude into the lumina of the ducts, they produce characteristic "filling defects" which can be readily detected. The technical simplicity and the diagnostic value of these mammograms can best be portrayed by the presentation of typical cases.

UNILATERAL CENTRALLY PLACED PAPILLOMA

Case 1. P. P., housewife, forty-four years of age, entered the hospital complaining of a serosanguineous secretion escaping from the left nipple of six weeks duration. Any pressure or manipulation increased the amount of discharge. She had nursed each of her three children and had provided a bounteous supply of milk. Trauma and infection were not causative agents.

The breasts were small, atrophic and presented multiparous characteristics. There were no palpable tumor masses or points of localized tenderness. The subareolar tissues felt normal but slight pressure expressed a serosanguineous discharge from one of the centrally placed milk ducts and a clear serous fluid from the others. Transillumination and soft tissue roentgenograms did not outline the tumor or show existing abnormalities. The only pathological sign was the serosanguineous discharge which escaped from an apparently normal breast.

The nature of the discharge, the absence of a palpable neoplasm and the age of the patient suggested the diagnosis of a ductal papilloma. This, however, was merely conjectural. Having thus far failed to visualize the offending neoplasm, contrast mammograms were employed. The nipple was thoroughly cleansed with alcohol, not only to render the field aseptic but to remove any incrustations or plugs of inspissated material which might be obstructing the orifices of the lactiferous ducts. A gentle stripping massage expressed one drop of bright red blood. The offending estuary was cannulized by a blunt No. 26 gauge needle and 2 c.c. of thorotrast were introduced. The patient was immediately placed in a lateral recumbent position with the injected breast supported by a Meyers angle board and a lateral stereoscopic roentgenogram was made. The diseased duct and all of its ramifying lacteals were graphically outlined. The portion of the duct traversing the nipple was smooth, regular and of a uniform diameter. The ampullic segment, however, contained a definite "filling defect" which was interpreted as being a tumor displacing the

contrast medium (Fig. 1. A). The neoplasm was exceedingly small, well circumscribed and had not destroyed the ductal continuity. Dr. H. B. Hunt made the roentgenographic diagnosis of a benign papilloma. The lacteals and acini appeared normal.

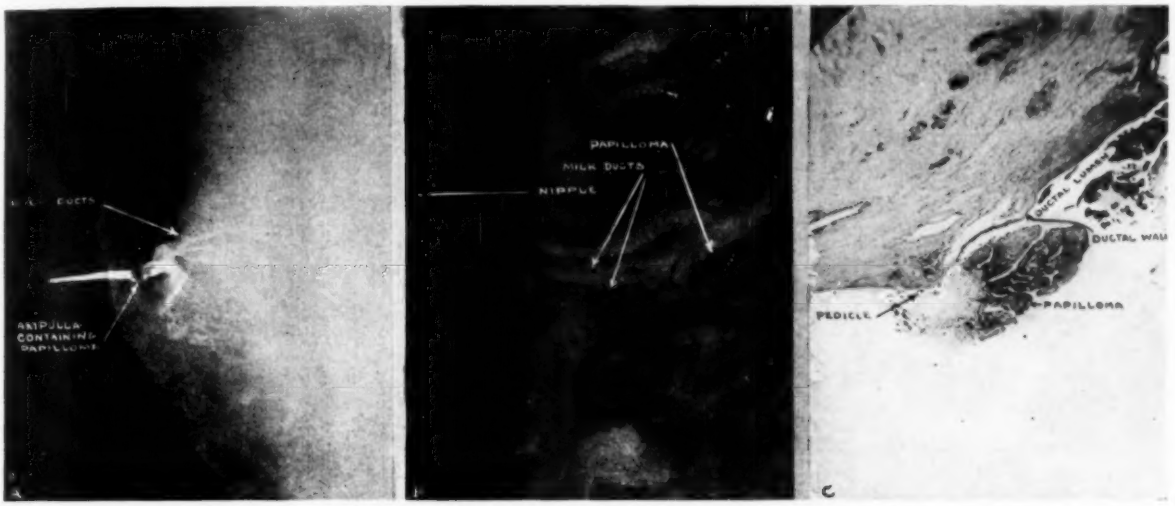

FIG. 1. Case 1. A, 2 c.c. thorotrast injected into the bleeding duct; the "negative shadow" indicates a tumor protruding into the ductal lumen; B, photograph shows the papilloma in the duct; C, photomicrograph of the papilloma showing its attachment to the duct wall.

In order to excise the papilloma it would have been necessary to destroy the continuity of the regional ducts, therefore, a simple mastectomy was performed. A small papilloma, about 0.25 cm. in diameter, was found arising from the wall of a centrally placed milk duct (Fig. 1. B). Its base was not indurated nor was there any evidence of malignant transformation (Fig. 1. C).

UNILATERAL PERIPHERALLY PLACED PAPILLOMA

CASE II. L. McC., a twenty-eight year old housewife, was worried about her "bleeding breast." Three months before, while adjusting her brassiere, she had observed a drop of bright red blood seeping from the right nipple, but as there was no pain and no palpable tumor she ignored the incident. Two weeks later a similar discharge was observed but self-examination again failed to reveal any cause for concern. The bleeding increased in frequency until it became a daily occurrence. She finally consulted her physician who, being unable to feel any tumor or find any point of localized tenderness, told her that such troubles were "inconsequential." The hemorrhagic discharge, however, persisted. There was no history of injury, cracked nipple, "caked breast" or infection. She had nursed each of her four children, the youngest of whom was two years of age.

Both breasts were large, pendulous and obese. No tumefaction, ulceration or discharge from the nipple was seen. The milk ducts were distended but compressible. Even the most meticulous and painstaking palpation failed to locate the tumor and no point of localized tenderness was found. Pressure on the upper outer quadrant of the right breast expressed a dram of frank blood from one gaping estuary. The entire mammary gland was translucent and no semblance of a papilloma could be found either by transillumination or soft tissue roentgenograms.

An accurate diagnosis was impossible for the only abnormal finding was the bloody discharge from the nipple. Having failed thus far to identify the causative agent, mammographic visualization studies were employed, after introducing 4 cms. of lipoiodine into the bleeding duct. The resulting mammogram accurately visualized the diseased duct and all of its tributaries. The ampullic portion was dilated to five or six times its regular size while the terminal lacteals and acini appeared normal (Fig. 2. A). No "filling defects" were seen. Six other ducts were also injected and while their ampullae appeared to be slightly dilated, none of them had undergone such an extensive cystic degeneration as the one which emitted the bloody discharge. The localized cystic dilatation of the offending duct combined with the

hemorrhagic discharge suggested the diagnosis of a ductal papilloma.

A mastectomy was performed and the accompanying photograph shows the small papillomatous neoplasm which was projecting into the dilated ampulla (Fig. 2. B). The duct did not contain any blood nor could a point of localized bleeding be found. Histologically, the tumor proved to be a benign papilloma (Fig. 2. C).

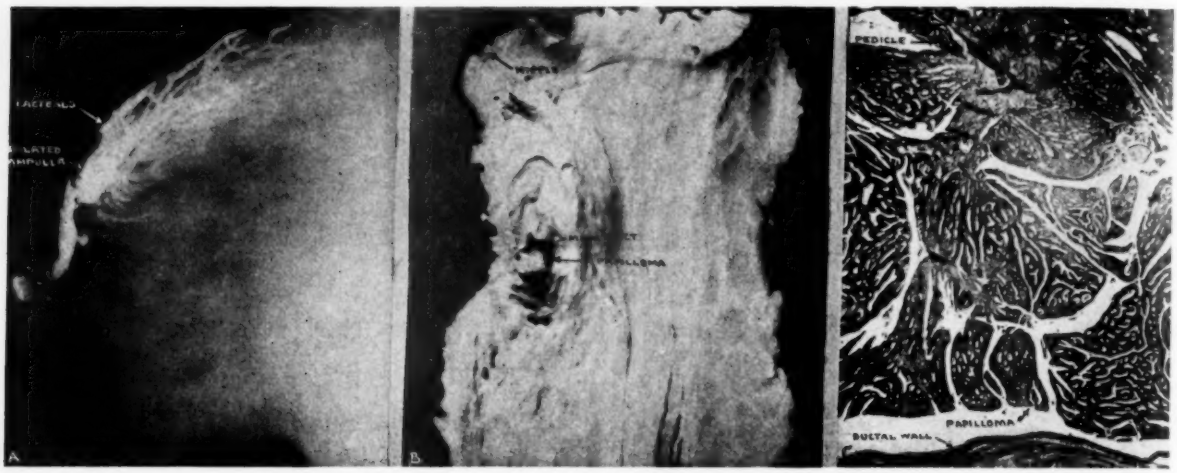

FIG. 2. Case II. A, 4 c.c. lipoiodine injected into the bleeding duct; note the dilated ampulla and branching lacteals; no negative shadow was seen; B, the papilloma can be seen protruding in the ductal lumen; C, histologically the tumor was a benign papilloma.

This case presented an interesting diagnostic problem. Why did this particular papilloma fail to produce the characteristic "filling defect" which was observed in the previous case? The answer is obvious. Lipoidine is such a dense radiopaque material and has such a high viscosity that it forms an impenetrable covering around the tumor and thus obscures it. For this reason, thorotrast is a much better mammographic agent.

MULTIPLE AND BILATERAL PAPILLOMATA

CASE III. R. S., female, stenographer, forty-one years of age, complained of a "bleeding breast." On several occasions during the previous three years she had observed a few drops of blood-tinged fluid escaping from the right nipple. Frequent self-examinations failed to demonstrate any abnormalities so she thought the bleeding was caused by the incipient menopause. Two weeks before admission, however, she grew alarmed for the bloody discharge became persistent. She immediately consulted her physician but he was unable to determine the cause of the bleeding.

On examination both breasts were found to be small, soft and spongy. In the outer lower quadrant of the right breast a small compressible tumor could be felt. When pressure was exerted on this cystic mass, the regional ducts became tense and distended but no secretions were expressed from the nipple. While examining the upper medial quadrant of the same breast, a few drops of fresh blood escaped from a peripherally placed duct. No tumor mass or area of localized tenderness could be palpated. Transillumination and soft tissue roentgenograms again failed to demonstrate any abnormalities. The left breast, as well as the right, was asymptomatic and appeared to be normal.

In view of our previous experiences, it was believed that the cause of the hemorrhagic discharge could be determined by contrast mammograms. The bleeding estuary was readily cannulized and 1 c.c. of thorotrast was introduced. The roentgenogram gave a vivid anatomic pattern of the injected ducts. The dilated ampulla was distorted by a large filling defect. This deformity was interpreted as being a pedunculated papilloma protruding into the ductal lumen. In order to preclude the possibility of an artefact, such as an air bubble, an additional 3 c.c. of thorotrast was injected into the same duct. The contrast medium was forced beyond the obstructing tumor and entered the terminal ducts and acini. Not only was the ampullic tumor still visible but a second and similar deformity was ob-

served at the junction of two smaller tributaries (Fig. 3. A). The diagnosis of multiple papillomata was very evident.

a "negative shadow" or "filling defect" could be seen. It maintained a constant size, shape and position in subsequent mammograms and

Fig. 3. Case III. Right breast. A, bleeding duct injected with 1 c.c. thorotrast; observe the two "negative shadows" in the ducts; B, the offending duct contains two papillomata; C, the papilliferous tumor practically occludes the ductal lumen; benign papilloma.

Hoping to familiarize ourselves with the ductal pattern of a normal breast, contrast mammograms were made of the asymptomatic left mammary gland. Four centrally placed lactiferous ducts were cannulized and injected with 10 c.c. of thorotrast. The stereoscopic mammogram revealed some startling and com-

was interpreted as being a pedunculated papilloma arising from the walls of a preformed cyst.

After making the mammographic diagnosis of multiple papillomata in one breast and a large cyst undergoing papillomatous transformation in the other, a bilateral mastectomy was performed.

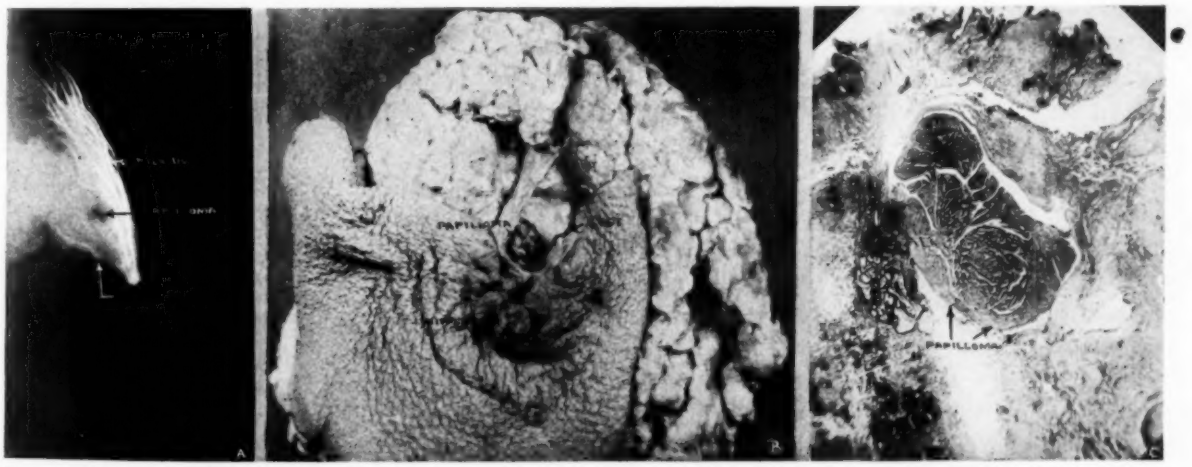

Fig. 4. Case III. Left breast. A, thorotrast outlines a large cyst which contains a "negative shadow"; B, parent cyst and pedunculated papilloma are exposed; C, photomicrograph of benign papilloma.

pletely unexpected findings. The contrast fluid had entered a large cystic cavity which was formed by a confluence of the injected ducts (Fig. 4. A). The delimiting wall of the cyst and the communicating regional ducts were clearly visualized. In the central portion of the cavity

The accompanying photographs verify the mammographic diagnosis. In the right breast the ampullic papilloma was soft and friable (Fig. 3. B). There was one small hyperemic area which was covered with a recently formed blood clot, obviously the site of hemorrhage.

The tumor mass was attached to the duct wall by a broad non-infiltrating base. Two centimeters farther along this duct, at the junction of two smaller branches, was a kindred tumor which protruded into both ductal lumina so as to completely obstruct them. No evidence of malignancy was found (Fig. 3. C).

The left breast also presented some interesting findings. In the subareolar area was a dilated cystic cavity which communicated with several adjacent ducts, and arising from its wall was a small papilliferous tumor which projected into the main cavity (Fig. 4. B). Obviously this neoplasm originated from the wall of a preformed cyst. The papilloma was soft, friable and spongy and there was no evidence of intracystic or intraductal hemorrhage. Such an observation is interesting for this tumor was about three times larger than the bleeding neoplasm of the opposite breast. Its histological structure was that of a benign papilloma (Fig. 4. C).

CONCLUSIONS

Certain generalizations can be drawn from these studies.

1. Bleeding from the nipple constitutes a distinct warning that some profound pathologic change has taken place in the breast. It may announce the presence of carcinoma, papilloma, Paget's disease, cysts, infections, traumatic degeneration, or vicarious menstruation.[5] Inability to determine the exact cause affords no license for passive conservatism as incurable sequels may follow.

2. A hemorrhagic or serosanguineous discharge from a breast in which no tumors can be palpated is most likely to be caused by a duct papilloma. These tumors are soft, compressible and spongy and hence cannot be readily discerned by palpation. Positive findings are valuable, negative ones are meaningless. In none of our patients were the neoplasms palpable in spite of the fact that one was as large as a marble.

3. Papillomatous tumors are often multiple. They may arise in various segments of the same duct or in other ducts of the same or opposite breast.

4. The size of the papilloma bears no relationship to its symptomatology. A small pinpoint tumor may bleed rather profusely (Case I), while much larger ones may be asymptomatic (Case III).

5. Papillomatous tumors usually cannot be visualized either by transillumination studies or by soft tissue roentgenograms. The neoplastic tissues have the same translucency as the surrounding matrix and differentiation is seldom possible. Occasionally, a large intracystic hemorrhage will permit transillumination identification.

6. The absence of symptoms and physical findings is no assurance that papillomata are not present.

7. Papillomatous neoplasms can be readily visualized by contrast mammograms. The tumor produces a characteristic "filling defect" of the ductal lumen. In the 3 cases presented the papillomata were not palpable, nor were they seen on transillumination or soft tissue roentgenograms and yet they were clearly visualized by mammographic studies. Thorotrast is the most suitable contrast agent and has been used in more than 375 mammograms. The escaping discharge accurately locates the offending duct which can be easily cannulized and suitable quantities of thorotrast introduced. Any discharge from a nonlactating breast, whether serous, serosanguineous, hemorrhagic, purulent, or "milky," demands mammographic studies. Such pathological conditions as carcinoma, cysts, galactoceles, papillomata and breast abscesses have been visualized and diagnosed by contrast mammograms. While papilloma of the milk ducts is essentially a benign epithelial tumor, it may undergo carcinomatous transformation if permitted to pass through its evolutionary cycle.

8. These visualization mammograms not only possess great diagnostic value but they aid the surgeon in determining the proper type of corrective treatment. They accurately locate the offending tumor and thus indicate the breast lobule which is to be excised. Nothing is more embarrassing than to remove a segment of the mam-

mary gland and then have the pathologist report that no papilloma could be found. Multiple tumors can be recognized preoperatively, in which event a simple mastectomy replaces local excision of the neoplasm.

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Infectious diseases is one of the great tragedies of living things—the struggle for existence between different forms of life. Man sees it from his own prejudiced point of view; but clams, oysters, insects, fish, flowers, tobacco, potatoes, tomatoes, fruit, shrubs, trees, have their own varieties of smallpox, measles, cancer, or tuberculosis. Incessantly, the ruthless war goes on, without quarter or armistice—a nationalism of species against species.

Rats, Lice and History by Hans Zinsser.

LYNCH'S SIMPLIFICATION OF PERINEAL EXCISION OF RECTUM

PRELIMINARY REPORT

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NEW YORK

PERINEAL excision of the rectum for the eradication of carcinoma was first performed some two hundred years ago. Since then the modifications in technique have been legion. Nevertheless three great problems still confronted the surgeon performing this operation: (1) to find a suitable anesthetic, (2) to control the secondary hemorrhage, and (3) to avoid postoperative infection.

The first of these difficulties was overcome when low spinal anesthesia became established. Anesthesia so induced permits of ample time for the procedure without endangering the life of the patient. We have now devised an operative technique which appears to overcome the other two problems, hemorrhage and infection, which are in reality complementary. Our routine is as follows:

PREPARATION

The patient is prepared in the same way as for any major operation. The desiderata are that the subject shall come to the operating table in the best condition possible in the time circumstances allow. To this end attention is directed towards building up the hemoglobin, the body fluids and the salts; to protecting the liver with glucose; and to eliminating the toxins from the renal and intestinal tracts.

The lower bowel is emptied by means of castor oil administered by mouth in the morning, and by cornstarch enemas in the evening. It is important that no purge be given within forty-eight hours of operation as that interval is essential for the elimination from the kidneys of any toxins liberated by such medication. The last enema should be given the night before operation.

Preoperative Medication. A good night's sleep should be insured by the administration of a hypnotic. A full dose of morphine coupled with atropin grain $\frac{1}{150}$ should be given one-half hour before operation.

OPERATIVE PROCEDURE

Stage 1. Following low spinal anesthesia, with the patient in the left Sims position a pursestring suture is placed about the anus. An incision is made in the midline extending from the posterior margin of the anus to the sacrococcygeal joint. The coccyx is removed subperiosteally. All bleeding points are clamped and tied. The rectum is freed from the hollow of the sacrum by blunt dissection.

Stage 2. With the patient in the lithotomy position, the anus is circumscribed by an incision 2.5 cm. from its midline. The line of cleavage between the prostate or vagina and the bowel is defined, and the bowel pulled taut. The lateral ligaments of the rectum are isolated and incised between clamps. The peritoneal reflection is put on the stretch by gentle traction on the rectum and is pushed cephalad by blunt dissection. If it becomes necessary to open the peritoneal cavity, we never hesitate. All bleeding points are clamped and tied, and the wound thoroughly dried by means of very hot packs.

This procedure leaves the bowel entirely freed from all of its attachments, and the operator may deliver as much of it as is considered necessary. A crushing clamp is applied at the optimum point, above the growth, and the caudad bowel is excised with the cautery. This clamp is now removed and a rather soft rubber tube, 2 cm. in diameter, is inserted into the bowel and

secured with one or two interrupted sutures. The wound is then swabbed with a 10 per cent solution of tannic acid in alcohol, following which the cavity is lightly packed with a roll of 9 inch gauze moistened with the same solution. The rubber tube is cut off at the level of the buttocks. The packing is held in place with a T-binder, and the patient returned to bed.

POSTOPERATIVE CARE

Postoperatively the patient is treated as if in shock, since it is much easier to hold a toboggan at the top of the hill than to arrest one in flight. The foot of the bed is raised on blocks, an electric cradle is placed over the patient and a continuous intravenous saline drip is commenced. Morphine and coramine are given as needed. The blocks are removed at the end of twelve hours. The packing is kept moist with the 10 per cent solution of tannic acid in alcohol and is removed at the end of forty-eight or seventy-two hours, replacing it with sufficient gauze, moistened in like manner, to keep the two sides of the wound from touching. Mandellic acid is given as a prophylaxis against urinary infection, but must be administered circumspectly.

The rubber tube ordinarily is found free in the dressings on the fourth day. Failing in this, it should be removed on the fifth postoperative day.

No effort is made to stimulate or retard defecation until the fifth day, at which time if the patient's condition warrants, a dose of castor oil is given by mouth one hour before breakfast. From this point on every effort is made to prevent constipation.

We have followed this routine with our last 15 cases. On the average they were able to get out of bed on the tenth day following operation, and left the hospital during the third week. So far there has been only one death, and that due to a cardiac condition. We fully realize that this series is insufficient to permit of an estimate as to the true mortality under this technique. Our experience has convinced us, however, that there is less risk attached to the procedure than pertains to any of the others which we have adopted.

CONCLUSIONS

Briefly, this modification of the usual technique consists in (1) leaving the wound wide open; (2) excising the bowel at the new level of the peritoneal reflection, with no attempt to anchor the bowel to it; and (3) in dressing the wound with a 10 per cent solution of tannic acid in alcohol. From these changes the following benefits are derived:

1. Leaving the wound wide open eliminates the danger of the formation of pus pockets.

2. The absence of sepsis obviates the danger of secondary hemorrhage.

3. The 10 per cent solution of tannic acid in alcohol forms a coating which is strong enough to prevent saprophytic infections, but is not dense enough to allow virulent organisms to grow under cover of it.

4. Excising the bowel at the new peritoneal reflection lessens the danger of peritonitis and prevents the tendency to prolapse which is so frequently attendant upon this type of operation.

5. This simplification of the technique greatly lessens the operating time.

6. The formation of the tannic albuminate insures a healthy granulating wound and thereby diminishes the period of hospitalization.

7. The final cosmetic result compares very favorably with any operation that has as its object a perineal stoma.

SIMPLE TECHNIQUE FOR CECOSTOMY*

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THE type of colostomy that one makes depends on the indications presented, the conditions under which it must be performed and on the anatomical variations of structure which are encountered. In most elective colostomies one has a choice of several fairly standard operations. The majority of these are designed with the idea of facilitating subsequent operative procedures. It has seemed to us that considerable confusion has resulted from the use of the words "permanent" and "temporary" in designating different types of operation.

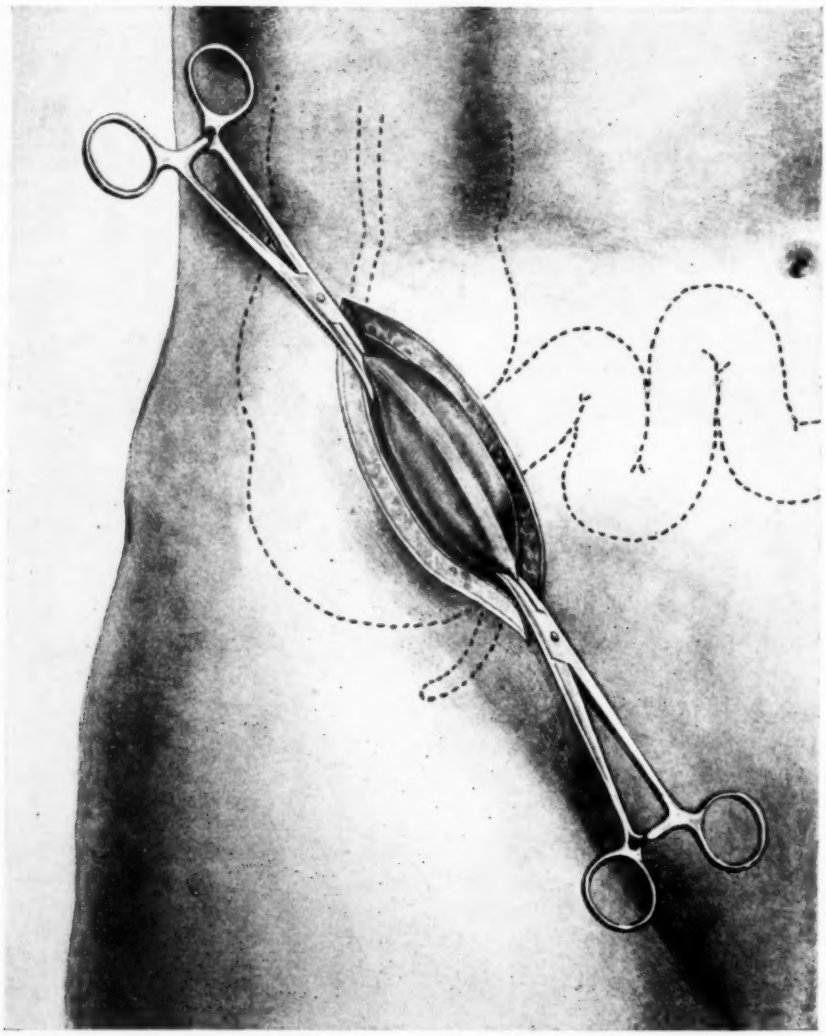

FIG. 1. Portion of cecum isolated and fixed to wound with curved forceps.

We prefer to divide them into two principle groups:

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1. Colostomy in continuity; no spur or interruption in the continuity of the bowel.

2. Complete colostomy; spur present or complete interruption in continuity of bowel.

Colostomy in continuity is indicated when marked distention of the large bowel has occurred and the causative obstruction is so complete that no relief can be obtained through the natural way by any means. The dangers incident to prolonged, increasing distention must be avoided by early relief. The primary consideration is to relieve the distention and prevent further distention. The secondary consideration is to provide a vent through which the fecal current can overflow. No particular attempt is made to divert the fecal stream away from the site of obstruction. It is not uncommon for the obstruction to open after the relief of distention and for the feces to again proceed through the natural channel.

A complete colostomy is one in which the fecal stream is diverted through an opening on the surface. None of the fecal current is permitted to pass into the distal segment of bowel from which the proximal segment is separated. Different means have been employed to prevent this, in some methods the stoma of the distal segment is closed by mechanical means while in other cases it is placed at such a distance as to make it impossible for the feces to find their way into the distal stoma.

The location of the colostomy must be proximal to the site of the obstruction. Thus it may be made in any accessible portion of the large bowel. As a rule, the more distal the site of the stoma and the closer to the obstruction, the more effective it becomes. Where obstruction is present and associated with marked distention, a stoma in the right half of the colon, preferably the cecum, is usually most suitable. The right half of the colon usually has liquid fecal matter and the left half usually has formed fecal matter, the consistency becoming firmer in the descending colon, sigmoid and rectum.

In some cases of marked obstructive distention from lesions of the sigmoid or rectum, it may be desirable to perform a cecostomy for purposes of decompression. Later a left iliac colostomy may be made since it is more desirable as an artificial anus. The cecostomy may be left to close spontaneously or may be closed by operation.

Fig. 2. Isolated portion surrounded by iodoform gauze, A, placed between cecum and parietal peritoneum.

A type of colostomy which has served us very efficiently in a series of difficult cases in which the patients were markedly distended and in whom the intestinal canal could not be decompressed by any of the methods of suction or irrigation will be described. It is also especially useful in the obese individual who is markedly distended and in whom the mesocolon is likely to be very short.

TECHNIQUE

Under local infiltration anesthesia the cecum is exposed through a muscle splitting incision similar to that used for appendectomy.

The anterior wall of the cecum is drawn gently through the incision and clamped along the anterior tenia with one or two

curved forceps (Fig. 1). No sutures are used anywhere in the operation.

Between the serosa of the cecum and the parietal peritoneum a strip of iodoform gauze is inserted to facilitate the formation of protective adhesions (Fig. 2).

When obstructive distention of the large bowel has been present for a long time, a plastic exudate commonly develops about the cecum which may eventuate in adhesions between the cecum and the adjacent parietal peritoneum. If this has occurred, one may introduce immediately a cannula without danger of contaminating the general peritoneal cavity. In the usual cases the incision is packed open down to the peritoneum and left undisturbed for a period of twelve hours. A catheter then may be inserted into the lumen of the cecum by introduction through an opening made between the forceps or immediately below the forceps (Fig. 3).

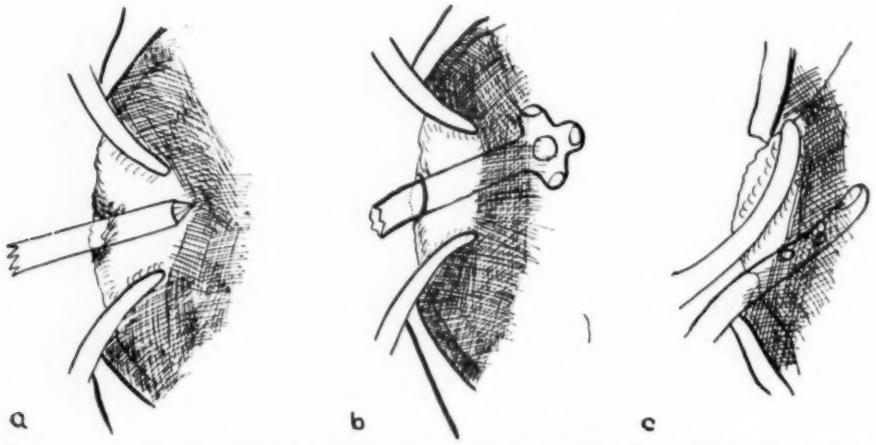

Fig. 3. *a*, trocar introduced after adhesions have formed between cecum and peritoneum, gauze not removed; *b*, Pezzar catheter in place; *c*, when only a small portion of cecum can be isolated and surrounded with gauze, a catheter may be introduced adjacent to the forceps.

SUMMARY

A method of cecostomy is presented which is simple and permits decompression in a patient who cannot be subjected readily to other more complicated types of operation. We wish to emphasize that this form of colostomy, or cecostomy, is not a substitute for any other method. It is suggested only for that group of cases which appear to be without recourse because of "mountainous" distention or obesity. No claim is made for originality but its proved value urges us to present it.

SHOCK

A STUDY OF THE PARTIAL AVAILABLE MODERN LITERATURE OF SHOCK

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ONE is tempted when undertaking a review of the literature of shock to enter his subject rather lightly and with at least as much assurance as he thinks he feels when called to meet its emergency in the surgery. However confident one feels in the early phases of the perusal, it is not long before a slow sense of consternation enters as one is deluged with conflicting theories, and data which refuse to integrate.

In reading the numerous articles one finds that there is no satisfactory account of shock, and he is led to believe that it is still a chaotic field where individuals, committees and commissions wander, while in the hospitals the emergency of shock is met daily in hard practice, now and then successfully. When sufficient time has elapsed, one realizes that a few well supported principles have appeared gradually, which seem well worth our search.

No attempt has been made to make a complete survey of the literature; we have confined ourselves to a review embracing the literature available through a fairly comprehensive list of libraries. We have rarely expressed our own opinions, either those which might be derived from experimental work or those acquired through clinical experience. Both principles and emphasis have been left largely to the actual content of the literature as it exists.

On the completion of this fairly long review specific acknowledgment is made each library mentioned at the end of the paper other than the author's own. This particular acknowledgment is extended also to each author quoted and each publisher mentioned in the reference list. Acknowledgment is also due my secretary, Myra Keplinger, A.B., for her assiduous help in abstracting and collating the materials with which we have dealt.

Definitions of Shock. Webster[1] defines shock by the use of words such as "collapse" and "concussion." Roget[2] at greater length uses "attack, paroxysm, seizure, apoplexy, paralysis; thrill, excitement, agitation; concussion, crash, collision, impact; blow, ordeal."

We at once sense in a reconsideration of these terms that the word *shock* has come in the field of medicine to have special significance, one not conveyed usually by such terms as "collapse" or "concussion." It has, in other words, become a technical term and has acquired somewhat exact limits to its meaning.

Dorland[3] describes shock as a

> sudden vital depression, due to an injury or emotion which makes an untoward impression upon the nervous system. Shock may be slight and transient, or profound and even fatal. In severe shock the prostration may amount to immobility or syncope; but in other cases the patient is restless and excited. Recovery from shock is followed by more or less quickening of the pulse and respiration and abnormally high temperature.

Schorcher[4] defines shock as

> a clinical state resulting from a sudden trauma of the nervous elements influencing the central nervous system by way of the reflex arc. It is accompanied by a rise in the blood pressure followed later by a fall. Collapse, on the other hand, while clinically similar to shock, differs from it causally in that it is the result of a chemical action. It is caused by the absorption of toxic substances and their effect primarily in the circulatory system. It is slower in development and is characterized by a primary fall in the blood pressure.

Rutherford[5] speaks of shock as "an interference with the most fundamental mechanism of life—an interference with

the mechanism for the transformation of energy."

Bickham[6] defines shock as

a reflex depression on the vital functions due to bodily injury, traumatic or operative, due to prolonged operation, or error of anesthetization, due to psychic influence, especially strong mental emotions of a distressing nature, or due to any other cause resulting in the exhaustion of inhibition of the vasomotor mechanism.

According to Wright[7] in his discussion of traumatic shock,

"Shock" is a condition in which the sensory and motor parts of the reflex arc are paralyzed to a greater or less degree, together with a profound disturbance of the circulation, subnormal temperature, and usually frequent shallow beathing; it may be seen typically following severe injuries.

According to Sherrington,[8]

The whole of that depression or suppression of nervous functions which ensues forthwith upon a mechanical injury of some part of the nervous system and is of temporary nature may be conveniently included as "shock." Goltz considered it entirely a collection of inhibition phenomena.

In a dissertation on operative shock Rehn,[9] Professor of Surgery at the University of Freiburg, Germany, says,

All of these considerations have served to contribute to the conception of shock the idea of a sudden occurrence precipitated in one way or another by some factor acting from without and followed by severe circulatory manifestations of a peripheral and central nature together with disturbances of other vegetative centers.

Definition of Shock Quoted from Early Literature. In Mann's[10] consideration of "The Peripheral Origin of Surgical Shock," he includes a brief historical review of the subject, quoting early definitions of "shock" by various authors. An example follows: "Travers ('An Inquiry Concerning Irritation,' London, 1826) states that 'shock is a species of functional concussion by which the influence of the brain over the organ of circulation is deranged or suspended.'"

Analogy between the Chemical Pathology of Cholera and Shock. With reference to the history of the subject of shock, Atchley and Loeb[11] say "In 1831 Dr. W. B. O'Shaughnessy of Newcastle-upon-Tyne wrote a brief letter to the London Medical Gazette which embodied the results of several years of what he termed his 'experimental inquiries' into the cholera." These authors

have taken time to trace the development of the chemical pathology of cholera because it presents probably the earliest instance in which there was an understanding recognition of the physiologic processes involved in the development of this common complication of many disease conditions, viz., dehydration, salt depletion, and shock. . . .

Chronologically, the next correct appraisal of the fundamental problem may be found in the Guy's Hospital reports of 1874. Dr. C. Hilton Fagge described, therein, "A Case of Diabetic Coma, treated with partial success by the injection of a saline solution into the blood." The most impressive feature of this pioneer experiment in therapy was the rationale which he offered. . . What suggested to my mind the advisability of injecting a saline solution into the blood in this case was the idea that coma was due to the drain of water from the system, caused by the diabetes. I suppose that the hypothesis upon which I acted was essentially similar to that which formed the basis of the like treatment in the collapse of cholera.

Characteristics of the Shock Syndrome as Recognized in Modern Surgical Practice. Orr[12] states that "James Latta, in 1795, is said to have been the first to use the term '*shock*' to describe this condition," referring to a syndrome occurring in surgical practice. Today in the syndrome of surgical or traumatic shock, according to Cannon[13] the following characteristics are recognized, a single case illustrating several or all of them: low venous pressure; low or falling arterial pressure; rapid, thready pulse; diminished blood volume; normal or increased peripheral red cell count and blood hemoglobin; leucocytosis; increased blood nitrogen; reduced blood alkali;

lowered metabolism; subnormal temperature; cold skin, moist with sweat; pallid, grayish or cyanotic appearance; thirst; shallow, rapid respirations; vomiting; restlessness; and anxiety progressing to mental dullness.

Symptoms and Signs Characterizing Shock Continued. According to Wright,[7]

> Breathing is characterized by long deep sighing respirations alternating with very superficial ones. The patient reacts very slightly and only to very painful stimuli. The limbs are toneless. Spontaneous movements are not made, but in response to urgent orders very brief limited movements may be executed. Complaint may be made of cold, faintness and deadness of all parts of the body.

Marshall and Piney[14] include in their enumeration of symptoms characterizing shock, "profound flaccidity of the muscular system."

Bickham[6] says,

> The pulse increases in rate, while decreasing in tension and volume—respiration increases in rapidity and shallowness—temperature is lowered, the pupils dilate . . . and in advanced shock nausea, hiccoughing, and vomiting may occur—incontinence of feces and retention of urine may be present—reflexes are lost—response to stimulation lessens—and coma and death supervene.

Crile[15] says,

> . . . whatever the cause of exhaustion (shock) certain basic phenomena are the same— . . . , diminished reserve alkalinity, and in acute phases, increased H-ion concentration of the blood; intracellular changes in the brain, the liver, and the adrenals; decreased electric conduction of the brain and increased electric conductance of the liver.

Moon[16] points out that "shock occurs not only following extensive surgery or trauma. It is seen in a wide variety of clinical conditions including extensive burns, poisoning with various substances, metabolic intoxications, abdominal emergencies, and severe acute infections."

Consideration of the "Constitutional Condition and Shock." The original subject for this paper was "The Constitutional Condition and Shock," which suggests primarily, consideration of the predisposing causes in the physical constitution which militate for or against the state called shock.

Are some body types more susceptible to shock than others? It seems a fertile subject for investigation but very little reference to it is made in the literature dealing with shock.

Crile[17] says in his lecture delivered before the Harvey Society in January, 1908, "It is axiomatic to state that the better the physical condition the less the shock."

Factors Predisposing to Shock. Rutherford,[5] discussing the significance and management of shock, lists the following predisposing causes of shock:

> 1. *Age:* The aged do not endure shock well. The risk is determined, not by the age of the patient but by the age of his circulatory apparatus. When aged patients are subjected to injury, the immediate circulation and other changes may be slight; but, under these conditions, the power of compensation or rebound is likely to be equally slight.

Mortimer[18] says with regard to the relationship of age to shock:

> Shock is more likely to occur in infants and young children than in adults, for the whole nervous system is more responsive to impressions, and diffusion of these readily occurs, as is found in the liability of infants to general convulsions from causes which do not have the same effects later in life . . .

Rutherford continues as follows:

> 11. *Sex:* Before puberty there is little or no difference in the susceptibility of the sexes. After puberty, especially after the menstrual period (begins), the female becomes less stable. The male grows more hardy. During the childbearing period the female develops an increased capacity for shock. During the menopause the female again becomes more susceptible to shock. After the period of developing menopause the female has entered upon an unbroken period of quietness and her resistance is increased. At the corresponding age, the male is carrying the greatest burden of his career,

and from this period on to the end he is not so good a surgical risk as is a woman.

III. *Time of Day:* There is a marked daily variation in the process of metabolism of the body, beginning in the morning and ending in the evening. The vital processes are more active, reserved forces are at a maximum, the psychic factor at a minimum on rising in the morning. The most unfavorable time is from one to two o'clock in the morning. Night accidents are likely to be more grave than equally severe ones occurring during the day. In lingering illnesses, more deaths occur after midnight than during the day.

Wakeley and Buxton[19] mention race in addition to age, sex, and time of day: "Race is of some importance; the colored races are not so easily shocked as the white races." These authors say also, "Nervous patients are notoriously bad subjects for operation. So-called 'psychic shock' may supervene in a patient anxious about an operation. This shock may even come on during the operation while the patient is still unconscious, and it certainly tends to be a real danger afterwards."

Among additional factors predisposing to shock Robertson[20] mentions the fact that "Injury to the liver by chloroform or phosphorus greatly reduces the power of an animal to withstand depletion of its plasma proteins."

Crile[15] says,

> We have found that exhaustion may be produced by an excess of, no less than by the want of thyroid or adrenal or brain activity. We have found that loss of liver function, want of oxygen, want of cardiac power, want of normal vasomotor action, of themselves, individually or in any combination, may predispose to or cause exhaustion (shock).

Wangensteen[21] includes "anemia, infection, malignancy, inanition, fever, obesity," as some of the conditions that predispose toward shock.

Varieties of Shock. There are several varieties of shock, depending upon the agents giving rise to the syndrome, and also upon the sequence of events in the subject exhibiting the phenomena. One has consequently:

1. Physical shock;
2. Chemical shock;
3. Electric shock;
4. Mental or psychic shock;
5. Shell shock;
6. Litigation "shock."

1. *Physical Shock.* Wound shock, surgical shock, and traumatic shock are examples of physical shock. Pleural shock, discussed, for example, by Hamilton and Rothstein[22] and also by Cocke[23] is to be mentioned as a subheading under traumatic shock. Hamilton and Rothstein define pleural shock thus: "Pleural shock (pleural reflex, pleural eclampsia, pleural epilepsy) is a syndrome caused by irritation or puncture of the pleura and is characterized by the appearance of a group of symptoms not unlike those of air embolism."

Obstetric shock, also, is a form of traumatic shock under the general heading of physical shock. Taylor,[24] discussing this form of shock, says "It is my growing belief that separation of the placenta, in whole or in part, is rapidly followed by the formation of histamine or allied substances . . . then a major fall of blood pressure takes place. This fall has Nature's desired effect of stopping further hemorrhage on a large scale. . . . "

Shock due to freezing is a form of physical shock, although the results of severe freezing which contribute to the shock state have been shown by Harkins[25] to be similar to those following severe burns which will be mentioned under chemical shock.

"Speed" shock is another variety of physical shock. It is the syndrome which has been described as resulting from rapid intravenous injections. Milbert,[26] quoting Hyman et al., says that these workers "are of the belief that a rapid injection of any molecule into the vein may result in widespread systemic disturbances . . . " Milbert's own conclusions, however, are "That the role of velocity does not play a clinically

perceptible part in administering fluids intravenously."

2. *Chemical shock* includes anaphylactic shock, insulin shock, colloid shock, etc.; but also, no doubt, is related in many of its ramifications to physical shock. Shock following severe burns may also be classified as chemical shock. Harkins,[27] considering experimental burns, writes in summary:

> . . . local accumulation of fluid in cases of burns . . . begins at the time of the burn and continues in the form of a decelerating curve until death. Accompanying the collection of fluid is a simultaneous increase in the concentration of blood as shown by an increase in the percentage of hemoglobin and in the hematocrit readings. After most of the fluid has accumulated, the fall in blood pressure sets in and continues rapidly until death occurs in a state of secondary shock.

Chemical shock is also probably related in many of its ramifications to the third division, i.e.,

3. *Electric Shock.* This variety of shock may easily be related immediately or remotely to both physical shock and chemical shock. According to Palfrey,[28] "The effect of electric shock is predominantly a paralysis of respiration." Among Pearl's[29] conclusions we find the following:

> (3) It is probable that death from electricity in higher animals is due, in a majority of cases, to primary fibrillation of the ventricles. In man this condition is hopeless unless prompt and heroic treatment is instituted. (4) Postmortem findings fail to explain the cause of electric death. Changes in the walls of the vessels are noteworthy. Heat rather than electrolysis is probably responsible for most pathologic changes . . . (10) The sequelae of electric shock are many and varied. The most important ones affect the skeletal and nervous systems.

4. *Mental or Psychic Shock.* "Psychic shock," says Mortimer,[18] "has been defined as the effect on the medullary centres of impulses from the centres of consciousness (pain, fear, and other emotions)." This type of shock obviously is or may be present in some degree in physical, chemical, and electrical shock, and may derive some of its reinforcements from any combination of them. It may, on the other hand occur spontaneously from purely psychic causes and then run off into phenomena identifiable as having physical, chemical, and electrical shock characteristics.

5. *Shell Shock.* This term, says Wiggers,[30] "is reserved in the literature for more distinctly nervous disturbances which more or less permanently disqualify the patient but do not lead to circulatory failure and death."

6. *Litigation "shock"* is a condition, says Fraser[31] (quoting McKendrick) which is

> incapable of medical definition or of proof or disproof. True shock is the result of excessive pain, of severe bodily damage or due to some very terrifying experience. Litigation or "nervous shock" as it is often termed, on the other hand is a condition that is not suddenly produced but has a gradual onset.

Somewhat obscurely, in a statement having possibly more legal than medical value, we read that Spilsbury[32] regards "'shock' . . . as one of the most overworked terms in the medico-legal vocabulary, as many deaths said to be due to shock should really be ascribed to such conditions as loss of blood, exhaustion, injury to a vital organ or concussion of the brain, etc."

According to its behavior in the animal organism, surgical shock is divided into two stages:

Primary shock is

> very frequently seen in those cases in which injury has been so severe that death is inevitable, or may quickly supervene, unless proper treatment is instituted at once. The symptoms of *primary* shock develop as soon as the wound is received, with the onset of pallor, profuse perspiration, chilly sensations and sometimes loss of consciousness. The pulse rate is increased, but the blood pressure reading may early be somewhat increased or normal.[12]

Cannon[13] observes that "*Primary* shock may be seen . . . either in the mortally

wounded, or after extensive hemorrhage, or in the relatively rare instances in which nervous elements seem to play an important role."

According to Mann and Essex,[33]

> There seems to be complete agreement that the primary feature, whether the shock is immediate or delayed, is low arterial blood pressure. In either type of shock, if a low blood pressure persists for an hour or more, the secondary condition appears and is attended by the phenomena associated with oxygen lack, such as decreased metabolic rate, and decreased alkaline reserve.

Speaking of primary shock, Friedlander[34] remarks that "it is altogether probable that as a consequence of wounding, there is a reflex relaxation of blood vessels similar to that which occurs in fainting." Wright[7] states that, "Early or primary shock, which sets in immediately following an injury . . . is probably due to afferent impulses causing reflex vasodilation and a profound fall of blood pressure."

Primary and Secondary Shock. "Recovery from primary shock may be quite prompt or it may develop into the secondary type."[12]

Wright[7] says secondary shock is "due chiefly to a capillary poison, probably histamine or some substance with a similar action which is liberated from the injured tissues."

Cannon[13] in this regard says: "The theory of a secondary shock which has the strongest support . . . is that of a toxic factor operating to cause an increased permeability of the capillary walls and a consequent reduction of blood volume by escape of plasma into the tissues."

In quoting Quenu, "who has also collected and summarized the studies of other French surgeons made during the war," Friedlander[34] says, "Secondary shock does not appear immediately after the reception of wounds. So that it is not of the nature of a nervous effect. The state is commonly well established before infection, and therefore is not of bacterial origin."

Cannon[13] says further:

> Exposure to cold, lack of water, rough carriage and absence of splinting of broken bone have been recognized as circumstances favorable to the onset of the symptoms. A still later aspect of secondary shock is that which is attended by infection, especially with gas bacilli.

Orr[12] says, "Secondary wound shock is the type usually encountered in the severely wounded. It is also the type frequently seen following serious or prolonged surgical operations."

Primary and Secondary Forms of Shock Distinguished. Holt,[35] opening a discussion on traumatic shock at a recent meeting of the Section of Surgery of the Royal Society of Medicine, compared circulatory changes in neurogenic (primary) shock and traumatic (secondary) shock thus:

Secondary Shock (Hematogenic)	Primary Shock (Neurogenic)
Blood pressure maintained.	Fall of arterial pressure.
Rise in pulse rate.	No rise in pulse rate.
Vasoconstriction.	Vasodilation.
Diminution in blood volume.	Blood volume not diminished.
O_2 consumption diminished.	No change in O_2 consumption.
Fall in blood pressure serious.	Fall in blood pressure not serious so long as respiratory center maintained.

New Descriptive Terms Suggested to Replace "Primary" and "Secondary" as Applied to Shock. He thought

> . . . that some other classification than the division of shock into *primary* and secondary was necessary. American writers had suggested the following classification: *hematogenic*, resulting from loss of circulating blood volume (muscle trauma); *neurogenic*, resulting from loss of central vascular tone (trivial injury, emotional distress); *vasogenic*, resulting from loss of peripheral vascular tone (histamine and other drug reactions). The adoption of some such classification would do much to remove the present confusion in which the pathology of this subject had been placed.[35]

Is There Such a Condition as "Secondary" Shock? At the same meeting at which these recommendations were made, it is noted that O'Shaughnessy declared "he had never seen a case of secondary shock nor read a satisfactory account of such a case, nor seen it experimentally produced." According to his experience, "following trauma the syndrome progressed either to recovery or to a fatal end. There was never an initial recovery followed by a later onset of shock."

OPINIONS REGARDING CAUSATION OF SHOCK

1. *Accumulation of Blood in the Capillaries.* Crile[15] uses the term "shock" to denote a state of exhaustion which has been developed rapidly by psychic, traumatic, toxic or thermal stimuli. Hewlett,[36] in resumé, says

> The marked hypotension of surgical and traumatic shock is not due in the main to a loss of arterial tone or to a failure on the part of the heart. It is due to a lessened flow of blood to the right auricle, caused mainly by an accumulation of blood in the smaller venules and capillaries. This accumulation may be due to a direct injury of the part or it may be caused by vascular poisons absorbed from the damaged tissues. There is no good evidence that nerve lesions play a dominant role in the pathogenesis of the more serious forms of surgical or traumatic shock.

2. *Loss of CO_2 from the Blood.* In discussing the causation of shock, McDowall[37] says,

> Of special importance in relation to central failure as a cause of surgical shock is undoubtedly sensory stimulation. . . .
>
> The real explanation has . . . been shown by Yandell Henderson to be due to the washing-out of carbon dioxide brought about by the hyperpnoea occasioned by the sensory stimulation. The acapnia or loss of carbon dioxide . . . brings about a reduction in the tone of the vaso-motor centre . . . In such anesthesia (light ether), as pointed out by Henderson, it is important to remember that apart from the fall in blood pressure, there may be brought about death from failure of respiration as a result of the washing-out of the normal stimulus—carbon dioxide. The amount of this gas in the blood becomes insufficient to stimulate the partly anesthetized centre and respiration becomes inadequate to supply the oxygen requirement of the body, especially of the heart and the medulla.

3. *Role of Anesthesia as a Causative Factor in Shock.* Statements by Knoefel[38] with reference to the role of anesthesia as a cause of shock may be quoted here. He says in part:

> . . . the secondary or delayed shock that may be produced by prolonged anesthesia with ether or chloroform is due to a concentration and reduction in volume of the circulating blood resulting from general stimulation of the sympathetic nervous system including the outpouring of adrenin . . . Banerji and Reid have just shown that in the hyperglycemia caused by ether and chloroform, the adrenals are importantly concerned, and have again demonstrated that such production of hyperglycemia is inhibited by amytal.
>
> Does amytal, with related substances prevent the general hyperactivity of the sympathetic nervous system that ether and chloroform produce? If they do, and there is some evidence for such a conclusion, then their use as basal narcotics receives important support in that the tendency of such volatile anesthetics to produce shock may be markedly counteracted.

Local Nerve Block in Shock Prophylaxis. The utility of local nerve block in prophylaxis of shock is mentioned by certain workers. Jones[39] declares that "Local nerve block with a cocaine derivative (nupercain) lessens damage to the central nervous system, allows relaxation with a minimal anesthesia, and prolonged freedom from pain in the postoperative period."

4. *Precipitation of Particles in Capillaries, Lymphatics, etc.* McDonagh[40] makes the following assertion:

> Shock is due to the precipitation of the protein particles of the plasma in the perivascular lymphatics, capillaries, and occasionally the arterioles of an important viscus. The particles are precipitated because they have become too large or too agglutinated to

circulate, therefore any agent that can cause these two physical changes to occur in excess may produce shock. The chemico-physical changes the protein particles in the plasma undergo to cause disease, are the same whether they be caused by a parasitic or a chemical invasion . . . When protein particles are dispersed the particles become smaller, more numerous, their Brownian movements increase, and their negative electric charge becomes more pronounced. Excessive dispersion results in intra-vascular clotting and causes one of the forms of shock. Experimentally, this form of shock is occasioned best by an overdose of para-thyroid extract.[40]

THEORIES OF SHOCK

The following theories of shock are those having support of the majority of investigators dealing with the subject:

1. "Acapnia" (Henderson).[41]

The fundamental condition (in the development of shock) is a disturbance of the respiratory metabolism of the tissues which leads, through processes which are still obscure, to the acapnial state. That this state may properly be defined as acapnia is proved by the fact that, given an adequate oxygen supply and a restoration of the hemoglobin to transport it, no other known procedure brings all the functions of the body back to their normal state so effectively and so rapidly as does inhalation of carbon dioxide.

2. "Acidosis," or hydrogen-ion increase in the blood. Cannon[13] says of the "acidotic" process: "Non-respirable acid, such as lactic acid, develops in the tissues because of oxygen-want and, uniting with the Na_2 of Na_2CO_3, drives off CO_2 which is breathed out."

3. "Adrenal." Theories on the basis of an "adrenal" factor are apparently poorly supported. "Neither the suggestion of overactivity nor the suggestion of underactivity of the adrenals has sufficient evidence in its favor to warrant attributing shock to the adrenal glands," according to Cannon.[13] Wiggers[30] more recently has expressed a like opinion. Conclusions of other investigators who have experimented with animals with and without adrenal glands in a shocked state are quoted below.[86-88,etc.]

4. "Capillary congestion." Blalock[42] says of this theory: "Starling . . . thought the lost blood (in shock) was mainly in the capillaries of skeletal muscles . . .

"Erlanger and his associates . . . noted (in experimental shock) that the capillaries and venules of the intestinal villi were distended with solid masses of red corpuscles."

5. "Fat embolism." Quoting Moynihan,[43]

The presence of fat emboli in the lungs and other viscera of patients dying with all the symptoms of acute surgical shock has been reported by Bissell (1917), who also remarked upon the large amount of fat in the venous blood of such patients, as well as in the blood of patients with broken bones. The frequency of shock in the wounded among patients with broken bones, or with multiple wounds of the soft parts, was also pointed out by Porter (1916) (and also by Porter[44] in 1920) as evidence of the part played by fat embolism in the production of shock.

6. "Inhibition." Blalock[42] says of this theory:

It was developed by Meltzer before the reduction of the blood volume in shock was recognized. He noted that dissection of the skin of the abdomen caused an inhibition of peristalsis. . . . However, as Cannon has emphasized, the cessation of intestinal movements is not due to the inhibition of activity in the central nervous system, but rather to activity there.

7. "Local vessel injury with local loss of fluid." This theory is mentioned by Blalock,[42] who says,

Many different types of experiments have been performed and the results indicate that the loss of blood at the site of injury and not the absorption of toxins is responsible for the diminution in the blood volume and the decline in blood pressure following trauma to an extremity.

8. "Traumatic toxemia."

The present outlook, Cannon[13] says, seems to be that not only the shock following burns,

but also the shock consequent on severe trauma, is properly in the same category with other forms of general depression of bodily functions and defective circulation due to the setting free of toxic material in the body, and that the nervous factors, which for so long a time have been regarded as of primary importance, should be relegated in most cases to a secondary position.

9. "Vasoconstriction." Cannon's[13] criticism of this theory is as follows: "The chief defect in the argument (in favor of vasoconstriction as a cause of shock) lies in a failure to account for a primary vasoconstriction capable of inducing the effects which Malcolm[62] describes."

10. "Vasomotor exhaustion." Crile[15] says,

> We have found that excessive emotion, exertion, injury, infection, loss of sleep, hemorrhage, or the injection of acids, alike may cause exhaustion; and that, whatever the cause of exhaustion certain basic phenomena are the same—muscular and mental weakness; diminished adaptive metabolism; increased respiration; increased pulse rate; sweating; diminished reserve alkalinity and in acute phases, increased H-ion concentration of the blood; intra-cellular changes in the brain, the liver, and the adrenals; decreased electric conductance of the brain and increased electric conductance of the liver.

Medical Shock. Atchley[45] emphasizes the

> importance of shock as a fairly frequent and very vital complication of certain *medical* conditions. It can be seen that the internist may find two types of shock. There is *anhydremic* shock, dependent on simple loss of blood volume, an example of which is diabetic coma; and there is *toxemic* shock, dependent on possible capillary paralysis from some bacterial or protein substance, an example of which is vasomotor collapse in pneumonia.

This author[45] reports the case of an intravenous rattlesnake bite with subsequent shock and eventual recovery.

Moon and Crawford[46] report the case of a man exhibiting the shock syndrome in mercuric chloride poisoning.

Muller, Myers, et al.[47] describe shock symptoms which occasionally follow "the intravenous injection of arsphenamine and other drugs or serums."

Fatal Shock Following Intracutaneous Test with Horse Serum. Freedman,[48] in reporting a fatal case of acute anaphylactic shock following intracutaneous test for sensitivity to horse serum, reviews the literature of related fatalities. In his opinion "Fatal reactions following the subcutaneous, intramuscular or intravenous injection of horse serum comprise the largest group of such anaphylactic deaths. Fatal reactions following the intracutaneous injection of a protein, however, are extremely rare."

Gas Bacillus and Shock. Buerger[49] points out a practical point in that, "The early local signs of gas bacillus infection when deep seated may be mistaken for local shock, all the more so since the limbs, already in the condition of local shock, were found especially prone to this type of bacterial invader."

Further Observations Regarding Traumatic Toxemia. Number 8 of the theories of shock causation, i.e., that of "Traumatic Toxemia," may be elaborated somewhat. It is significant that surgeons who served on the battle front during the Great War observed that some toxic agent very definitely played a part in so-called "shock" cases. Thus in the special report of the Medical Research Committee, Dale, Laidlaw, and Richards[50] make the following statements:

> Shock, as produced by histamine, seemed to us . . . to be a condition in which the circulation failed, and the arterial pressure fell to a low level, in spite of arterial constriction, because the blood drained away into the capillary network and tended to stagnate there, instead of returning to the heart through the veins. And in seeking for an explanation of this condition it seemed to us that, while loss of plasma and consequent thickening of the blood, when they occurred, would accentuate the tendency of the blood thus to accumulate at the periphery, the essential cause must be a general loss of the normal tone of the

capillary walls. All observations on the living circulation supports the idea that only a small part of the capillary network is at any one moment functioning as a channel for the blood. . . .

Histamine Shock. . . . An investigation carried out by two of us[50] (Dale and Richards, 1918) . . . furnished us with positive evidence in favor of the view that histamine, and by implication the group of substances having the same type of action, owe their effect on the circulation, whether the evanescent depressor effect of small doses or the shock-like effect of large doses, to their power of obliterating the normal tone of the capillaries.

Over against this factual data we read reports exemplified by the following excerpts: Miller,[51] of Los Angeles, observes that "the classically accepted theory that the production of histamin in the tissues by trauma increases capillary permeability, permitting the escape of fluid from the vascular bed is now being questioned." He quotes results obtained by Freed[51a] who "has shown that rats which are very resistant to histamin, are not shocked by extensive trauma unless they have been previously adrenalectomized."

"Vicious Circle" of Causal Events in Production of Shock. Miller[51] considers the causal sequences of events in shock to be as follows:

> Dehydration produces increased viscosity of the blood which in turn produces anoxemia, already shown to give sympathetic stimulation. Sympathetic stimulation results in increased secretion of epinephrin, which increases the rate of the denervated heart; thus demonstrating that dehydration produces sympathetic stimulation. (Note former quotation concerning sympathetic stimulation by ether and chloroform—Knoefel.[38]) In shock the vicious circle is completed by the observation that prolonged sympathetic hyperactivity producing vasoconstriction reduces blood flow in large areas of the body, resulting in anoxemia and increased permeability of the smaller blood vessels with the loss of proteins and fluid.

We find Wright[7] quoting Cannon, and observing:

> (i) Histamine is present in all tissues and is readily liberated as a result of even trivial injuries. (ii) Many of the features of surgical shock are identical with those of histamine poisoning, e.g., the low venous return, the diminished cardiac output, the low blood pressure, the rapid pulse, the diminished blood volume, the increased viscosity of the blood and the rise in the haemoglobin percentage, visible distension of the minute vessels in the bowel and the increase in the weight of intestinal loops.
>
> Injuries which liberate quite small quantities of this capillary poison may induce shock in the presence of (a) an anesthetic which sensitizes the blood vessels to the action of histamine, (b) other complicating factors which of themselves tend to lower the blood pressure, e.g. (i) exposure to cold; (ii) lack of food and water . . . ; (iii) considerable sweating which for some unknown reason often accompanies severe injuries, causes further loss of fluid, and also tends to lower the body temperature; (iv) hemorrhage by itself, even if twenty-five per cent of the blood volume is lost, does not give rise to a permanent fall of blood pressure, but hemorrhage combined with injury produces shock.

Histamine-like Tissue Extract Called by Various Names. For many years numerous investigators believed that histamine or a histamine-like substance released in the blood stream has been an accompaniment of, and a causative factor in the production of (secondary) shock. We find in the literature different terms referring to this toxic tissue product. In the accounts of his researches Turck[52] has given the name "cytost" to the tissue extract derived from injured tissues. De Nito,[53] discussing blood pressure reducing hormones and protein shock, designates "lymphoganglin" which is believed by him to be formed in the lymph nodes as the most important "hormone" of this kind.

In Defense of the Role of Histamin Substance in Shock Production. Clark[54] says in his concluding paragraph of an article on surgical shock,

> Although it would now appear as a result of recent work that loss of fluid from and into the

injured area and not histamine is the cause of shock, it would be as well to remember, as Dale (1933) puts it, that "we should be unwise on the ground of negative results of experiments on the crushing of the histamine-poor voluntary muscles to discount altogether the possibility that irritative handling and exposure of abdominal viscera, for example, which are relatively rich in it, might liberate histamine into the circulation in shock-producing amounts."

Traumatic vs. Histamin Shock. Holt[35] believes

No observer has satisfactorily demonstrated the presence of any depressor substance in the venous blood from a traumatized area; . . . The circulatory changes of traumatic shock differ considerably from those produced by the injection of histamine or other depressor substances obtained from tissue extracts. Marked differences were found in the changes which followed traumatic and histamine shock respectively. The changes indicated . . . are best set out in the following tabular form:

Traumatic Shock	Histamine Shock
Diminished circulating blood volume.	No marked change in blood volume.
Diminished output of heart before arterial blood pressure falls.	Decline in arterial blood pressure before any diminution in cardiac output.
Removal of small quantity of blood causes death.	Much larger quantity of blood must be removed to cause death.
Marked pallor of viscera.	Congested appearance of viscera.
Lungs pale and heart containing very little blood.	Lungs congested and heart well filled with blood.
Very little blood when solid viscera sectioned.	Solid organs bleeding freely on section.

Experimental Surgery on Animals Tells Less than the Whole Truth When Applied to Human Beings. O'Shaughnessy and Slome,[55] discussing etiology of shock on the basis of experimental work, declare that "our first conclusion is that a toxemia due to the elaboration of histamine, or any other depressor substance manufactured in the traumatized area, plays no part in the syndrome of traumatic shock." Speaking of the variance of results of the experimental work of these investigators and that of Cannon, Miller[56] says, "As is so often the case, experimental surgery on animals tells less than the whole truth when applied to human beings, for in this instance it can take no account of that most important human factor, the psychological attitude of the patient."

Roome and Wilson[57] also record results of experimental work which lead to the conclusion that "The findings do not support the 'toxic theory' of the etiology of traumatic shock."

Injury of Certain Parts of the Body Causes Shock More Readily than Injury of Other Parts. With regard to traumatic shock, Wangensteen[21] asserts that shock follows injury of certain parts of the body more readily than others:

Fractures of the lower extremities, especially if accompanied by considerable bruising of the muscles, are very likely to be accompanied by shock. . . . Fractures of the lower extremity are more frequently followed by shock than similar injuries in the upper, accounted for largely by the presence of larger muscles in the lower extremity giving rise to more bleeding when injured. Fractures of the femur and pelvis are frequently attended by shock.

Head injuries, unless accompanied by a good deal of external hemorrhage, rarely give rise to shock. Fractures of the skull, unless associated with intraventricular hemorrhage or injury of the medullary centers, rarely give rise to depression of blood pressure or shock. . . . Isolated fractures of the spine rarely produce shock. And *in a patient with a fracture of the spinal column presenting shock, concommitant injuries elsewhere such as intraperitoneal damage or a broken femur, should be looked for.* (Italics ours.)

Wounds of the thorax in which multiple fractures of ribs occur may give rise to shock, owing to the formation of numerous hematomas. Severe shock may also follow the fracture of a single rib, in which the lung is punctured with the accumulation of blood in the pleural cavity. . . .

Intraperitoneal Injury and Shock.[21] Intraperitoneal injury, unless accompanied by severe

hemorrhage, does not give rise to significant depression of blood pressure. Contrary to general belief, the perforation of a hollow intraperitoneal viscus is not followed by shock with immediate reduction of arterial pressure. Collapse and disability, however, are the rule, owing to the pain produced by irritation of the parietal peritoneum.

Condition of Blood Vessels in Shock. With regard to the state of blood vessels in shock there is difference of opinion as to whether the vessels are dilated or constricted. Friedlander[34] declares that "the fall in blood pressure is due to dilatation (and congestion) of the large venous trunks of the splanchnic area with coincident medullary anemia." On the other hand Orr[12] says that "Wallace, Fraser, and Drummond, after operating upon many hundreds of patients with abdominal injuries in all degrees of traumatic shock, have not found any splanchnic congestion to exist."

Parsons and Phemister,[58] discussing experimental results, say, "We necropsied dogs which died as a result of traumatism of the extremities and found no dilatation or engorgement of the blood vessels of the intestines and other abdominal viscera. On the contrary the intestines were pale."

Hewlett's[36] conclusions regarding the mechanical factors which lead to the low arterial pressure of shock are that "although some reduction in arterial resistance and in cardiac efficiency may be present, the chief cause of the hypotension is withdrawal of blood from the circulation partly through the passage of plasma into the tissues but mainly through stasis of blood in capillaries and small veins." Buerger[49] quotes Dale, Cannon, and Hooker, who account for fall of blood pressure in shock as "due to the fact that capillaries become paralyzed and dilated with stagnation within them, thus rendering the arteriole contractions ineffective."

Blalock[59] says,

> The initial changes in blood pressure (in shock) are to be considered as secondary to (1) compensatory vasoconstriction, and (2) diminished output per beat. The minute output is usually diminished, but in those experiments in which tachycardia develops early the minute output may remain normal while the initial changes in blood pressure occur.

Cutler and Scott,[60] without reference to dilatation or contraction of vessels, say,

> It now seems clear that shock is but a symptom complex resulting from a rapid diminution in the circulating blood volume below the point of circulatory efficiency. When a certain point (the critical level) is reached, the low blood pressure and insufficient supply of blood acts in a vicious circle as an added injury to the capillary bed.

Rost[61] remarks that

> investigations of Mauthner and Pick have shown (that) there is a constriction of the blood vessels both of the liver and the intestines. During the contraction of the liver capillaries, the venous blood in the intestinal vessels is choked back. A constriction also occurs in the capillaries of the lungs. The blood pressure falls because the heart receives nothing to pump.
>
> . . . According to the opinion of Crile and Mummery the blood pressure sinks because of paralysis of the vasomotor center. But numerous investigations of other writers, such as Malcolm, Seelig and Lyon, Mann, have shown that the blood vessels in shock are not dilated but constricted.

Malcolm,[62] in setting forth his views as opposed to those of Crile in the matter of the condition of blood vessels during shock, declares:

> Crile's conclusion requires further proof . . . because the assertion . . . that a lowering of blood pressure in the carotids must depend on a relaxation of some part of the vascular system is open to question. It is, in my opinion, an altogether untenable proposition. It appears to be founded on the idea that a contraction of the arteries causes a rise of blood pressure and a relaxation causes a fall. But this is a very incomplete and inexact statement of the well recognized law that the small arteries exercise a regulating or stopcock action on the flow of blood to the tissues. When fully set forth an essential part of the

stopcock mechanism is that a contraction raises the pressure in the large vessels, but at the same time lowers the pressure in the smaller ones, in those beyond the point where the stopcock is supposed to act. Hence, if any vessel has its lumen sufficiently contracted, the blood pressure in it will be lowered. I believe that in this fact we have the true explanation of the fall of blood pressure in shock.

McDowall[37] says,

When the shock is due to capillary dilation as a result of toxins, the arteries are undoubtedly constricted, as has been emphasized by Malcolm. This has indeed been shown experimentally by the writer and may be taken as evidence of an attempt at compensation, such as we know occurs in hemorrhage.

According to Parsons and Phemister,[58]

Handy and Phemister found that re-injection or circulation by means of a vivi-perfusion apparatus of several cubic centimeters of slightly traumatized or slightly haemolyzed blood in the femoral artery caused vaso-*dilation* in the limb of a dog, but that these same amounts of severely traumatized or completely haemolyzed blood caused vaso-*constriction* when circulated in the limb. We have tested this further by the injection of completely haemolyzed or severely traumatized blood in small quantities (1–50 c.c.) into the left heart or first portion of the aorta, so that it reaches the general capillary bed in high dilution. It produces a sharp decline in blood pressure which with 25–50 c.c. may be as much as 70 mm. of mercury. But the recovery was always very prompt (one-half to two minutes) and when it was injected more gradually the blood pressure would return to the previous level before the injection was completed . . . It seems that passage of the damaged blood through the lung capillaries robs it very largely of its vasodilator property, and passage through the capillaries of a limb completely destroys it.

The cause of the vasodilation is unknown. It was thought that it might be due to a histamine-like substance liberated from the broken down cells, but this is not so, since, when the amount of histamine injected into an animal is increased, the fall in blood pressure becomes more marked and prolonged, whereas very large amounts of extensively haemolyzed or severely traumatized blood (200–400 c.c.) circulated through a peripheral artery do not lower general blood pressure at all and when circulated through a vein produce only an initial fall for one to two minutes. This parodoxical behavior when varying concentrations and amounts of damaged bloods are injected may be due in part or whole to physical chemical changes, not to a toxin acting on the cells.

The Cause of Experimental Vasodilation Is Unknown. According to Wakeley and Buxton,[19]

In shock the lips are pale, and the skin is cold, a state of affairs not at all suggestive of dilatation of the surface capillaries. The splanchnic area is quite large enough to accommodate a great deal of blood. Experimental production of splanchnic dilatation results in a fall of blood pressure. In shock, however, the practical observations of the surgeon show that no such dilatation occurs.

The Blood Vessels in the Intestinal Tract in Shock. The intestines are pale in shock, and not red and engorged as would be the case with splanchnic dilatation. The capillaries of the muscles are under different conditions from the capillaries of the viscera and skin. Their activity is dependent on the state of the muscle, and it is possible that the *loss of tone* in the muscles in shock allows of passive dilatation of a large mass of capillaries.[19]

Petroff, Filatov, et al.[63] conducted experiments with volume determinations of kidneys, spleen, hind legs, and brain which indicate that the introduction of hemolyzed or foreign blood causes a marked narrowing in the lumen of the renal and splenic blood vessels. The circulation in the brain and in the hind legs was affected only slightly. . . . These experiments demonstrated that the main alterations in the circulatory system in hemolytic shock proceed from the alterations in the walls of the arteries and the veins.

Wesselkin et al.[64] "have been able to substantiate the basic experiments of Hesse and Filatov as to the spasm of the renal arteries constituting a characteristic symptom of the acute stage of shock."

Deoxygenation of Body Tissues in Shock. MacFee and Baldridge[65] say,

> the essential fact of shock is deoxygenation of body tissues, occurring from impairment of circulation . . . (which) results from diminution of blood volume in circulation. This loss is due to stagnation of blood in the capillary areas and to escape of plasma from capillary channels. Hemorrhage and dehydration are frequent factors.

Blood Cell Counts in Shock. Cannon[13] and his co-workers at Bethune made blood counts in a series of 27 cases of severe traumatic shock. "All but eleven had a capillary count which amounted to 6,000,000 red corpuscles or higher, and in eight cases it was more than 7,000,000 corpuscles." Venous counts were also made at the same time, and

> from these considerations it seems evident that the difference between capillary and venous red counts varies roughly with the degree of shock, and, since the venous count is approximately at the normal level or below it, the difference is due to concentration of the blood or stagnation of corpuscles in the capillaries.

Wright[7] says, "The plasma bicarbonate content is lowered in shock; this has been attributed to anoxemia, which causes incomplete combustion of carbohydrates and possibly of fats in the tissues with the formation of acid products which pass into the blood."

Reduction of Alkali Reserve in Shock. Bayliss states that "a part of the normal bicarbonate content becomes neutralized by combination with some fixed acid (lactic, etc.) produced in the tissues on account of the defective oxygen supply." Henderson and Haggard attribute this to over-ventilation of the lungs by rapid breathing. Cannon states that "the reduction of the alkali reserve is an indication of a fundamental difficulty occurring in the body, namely, an insufficient oxygen supply."[12]

Continuing in Cannon's[13] own phraseology:

> The failure of delivery of sufficient oxygen to the brain is likely to affect profoundly the normal metabolism of nerve cells in particular, and to lead to a disturbance of their functions. It becomes a matter of importance, therefore, to know at what point in an impaired circulation the oxygen delivery to organs becomes inadequate.

Rost[61] says, "Cobbett and Valte . . . see an increased viscosity of the blood as the cause of shock. . . . Short could not demonstrate this condition." In a discussion of fat embolism in surgical shock, Moynihan[43] says, "That the presence of fat in the blood stream in shock greatly increased its viscosity was demonstrated by Gauss."

Viscosity of the Blood in Shock. Cannon[13] says the increase in the number of corpuscles per c.c. increases the viscosity of blood in shock. He says also that "another way in which the viscosity factor might become prominent is through concentration of the corpuscles due to slowness of the blood flow itself." (Work of Cohnstein and Zuntz and by Mall and Welch cited.)

H-ion Concentration of the Blood in Shock. According to Kilduffe,[66] "When the blood pressure is lowered by shock, there is a fall of *p*H, and the same is true of hemorrhage."

Blalock[42] disagrees:

> The fall in blood pressure precedes a significant increase in the hydrogen-ion concentration of the blood. It is only in the terminal stages of shock that the alkali reserve is markedly reduced.

Crile,[15] also, says that H-ion concentration of blood is increased "in every type of intense overwhelming activation—by intense exertion, by intense emotion, by intense trauma; it was increased also by inhalation anesthesia, by hemorrhage . . . (etc.).

Aub and Cunningham[67] conclude that

> 1. There is a markedly diminished oxygen content of the venous blood in experimental traumatic shock. This change occurs before the blood pressure falls to a shock level and

is still present after apparent recovery from shock. . . .

Are the Blood and Lymph in Shock Toxic? Elman and Cole[68] carried out animal experiments to discover if possible whether or not blood and lymph in shock are toxic. "Without describing in detail these experiments it was apparent that none of the injected material proved toxic; and, when it was, there was no essential difference over the controls."

Earlier investigators and advocates of the "traumatic toxemia" theory of shock would disagree with the foregoing statement on the basis of experimental work as well as clinical observation.

Orr[12] summarizes findings which "present rather strong evidence that following trauma there is an absorption of toxic protein material from the damaged tissues which is a factor in the production of the shock state."

The Factor of Toxins in the Production of Shock. In their "Note on Muscle Injury in Relation to Shock," Bayliss[69] and Cannon say in part: "And, just as in the animal experiments recorded above, the injured muscle would produce metabolites, which, on being absorbed into the blood stream, would indicate their presence by a decrease in the blood pressure, with other signs of shock."

Robertson[20] notes that "Incoagulability of the blood is one of the symptoms of profound anaphylactic shock. . . . "

Leucocytes in Shock. Other statements with regard to blood characteristics in shock follow: Orr[12] says,

> Following severe trauma a leucocytosis as high as 20,000, or even higher, may develop within one or two hours. This leucocytosis rapidly diminishes and usually disappears within forty-eight hours. The reason for this rapid rise in the white cell count is certainly not infection. It is apparently due to the absorption of traumatic products. These clinical observations do not correspond to the observations of Crile and Mann, who found a decrease in leucocytes in experimental shock.

Sugar Content of the Blood in Shock. With regard to the sugar content of the blood in shock, Cannon[13] says, . . . there is no lack of sugar in the blood; indeed, . . . the amount is actually above the normal (0.1 per cent). Furthermore, there appears to be no relation between the variations of the carbon dioxid capacity of the blood and the percentages of sugar."

Aub and Wu,[70] studying experimental traumatic shock, conclude: "1. Animals with marked muscle trauma but without true shock showed only slight changes in total non-protein nitrogen, urea, creatin and sugar in the blood. These constituents, especially the creatin and the sugar, rose markedly as shock developed. In control animals the determined constituents showed no appreciable change. . . . "

Scarpello's[71] researches induce him to conclude that

> . . . in traumatic shock the increase in the blood sugar is due chiefly to an alteration of the glucoregulatory functioning of the hepatic cells. It is not easy to give an explanation of this phenomenon. It is probable that various factors act on the liver and increase the glycogenic functioning. By way of a simple hypothesis, it might be assumed that the traumatic shock causes disturbances in the field of the sympathetic nervous system which in turn act on the hepatic cells and increase the glycogenolytic functioning. On the other hand, it is not impossible that the disturbing movements of the intestine cause an absorption by the portal system of toxic substances, which, as has been shown for various toxins (Delbet, Marmier), may stimulate the hepatic cells to an increased glycogenolysis.

Nitrogen Content of the Blood in Shock. Cannon[13] says, "By a chemical analysis of the blood of wounded men, Duval and Grigaut determined that, parallel with a diminution of the non-protein nitrogen of traumatized tissues, there was an increase of non-protein nitrogen and of residual nitrogen (i.e. total non-protein nitrogen minus urea nitrogen) in the blood." In his study of "Certain Aspects of the Metabolic Response to Injury," Cuthbertson[72] con-

cludes: "(7) The curves of urinary excretion of nitrogen and the basal consumption of oxygen are generally parallel."

The Basal Metabolic Rate in Shock. With regard to the basal metabolic rate in shock, Aub[73] says in conclusion,

> . . . (2) Experimental traumatic shock causes a marked fall in the rate of basal metabolism to 70 per cent of the original level. The degree of fall is dependent upon the severity of the shock produced. . . . (4) The effect of hemorrhage is not constant. It may temporarily lower, or have no immediate effect on the metabolic rate. (5) Recovery from shock after blood transfusion is usually associated with a prompt return of the metabolic rate to a normal level.

Systemic Effects of Shock. Regarding the systemic effects of shock, Atchley and Loeb[11] say,

> The most significant effect is the interference with tissue function which results from circulatory stasis and diminished blood supply. No organ shows this disturbance more strikingly than does the kidney. Renal function is compromised and anuria frequently results. This, in turn, definitely upsets the acid base equilibrium of the body and in the case of diabetic shock prevents the excretion of ketone bodies, thus adding to the seriousness of the disease state. The tissues of the central nervous system are also included in the general damage and the resulting pathology doubtless plays a large part in the terminal stages.

Shock and Hemorrhage. Reference may be made briefly to the distinction between shock and hemorrhage. Crile[17] writes as follows: "In the absence of a history of either trauma or bleeding, without evidence of free fluid in cavities, and without a blood examination, . . . we believe it (differentiation between shock and hemorrhage) cannot with certainty be done." Orr,[12] discussing changes in the blood picture in shock, quoting from Crile's work, states that

> there is either a slight or no fall or a rise in hemoglobin, and in hemorrhage there is at first little or no fall, but after the loss of a fourth, a sixth, or a tenth of the fatal amount of blood the hemoglobin begins steadily to fall. The red cell count follows rather closely the curve of the hemoglobin in both hemorrhage and shock. Crile found relatively slight change, sometimes a fall in the leucocytes in shock, and in hemorrhage in every instance a rising leucocyte count. He then concludes that repeated and accurate observations upon the blood picture may differentiate between hemorrhage and shock.

Blalock[74] quotes from an editorial in the Journal of the American Medical Association[111] which summarizes work by Moon and Kennedy:

> In shock the blood becomes more concentrated, as shown by specific gravity, hemoglobin, and erythrocyte count; following hemorrhage, dilution of blood occurs. In shock there is widespread capillary dilatation of the viscera, congestion accompanied by edema and petechial hemorrhages; following hemorrhage the tissues are anemic. The differentiation of hemorrhage and shock is of more than academic importance. It is well known that recovery follows the introduction of physiologic solution of sodium chloride into the circulation of patients suffering from the simple loss of blood. The futility of this procedure in shock has been proved beyond question. The increased permeability of the capillaries, which is a characteristic feature of shock, allows saline solution to escape rapidly into the tissues. Solutions containing acacia or dextrose have been found more effective than saline solutions but not so effective as transfusion of blood. Even the latter is ineffective in profound shock.[74]

Is Differentiation between Shock and Hemorrhage Possible? Archibald and McLean, quoted by Orr,[12] also observed "that in hemorrhage intravenous saline solution was helpful, but useless in severe shock." Macleod[75] says, "The diagnosis (i.e. between surgical shock and hemorrhage) is clinched by the effect of transfusion; the hemorrhage case quickly recovers whereas that in shock only slowly, if at all." According to Bickham,[6] "marked hemorrhage results in collapse, rather than in shock—although differentiation of the phenomena is not easily made." Cannon[13] is even less

discriminatory: "Shock is hemorrhage and hemorrhage is shock."

Mann and Essex[33] point out that

shock and hemorrhage are identical in one respect, namely, that there is a decrease in the amount of circulating blood in both conditions. Thus there is a similarity in the physiologic aspects of the two conditions, although the pathologic findings may be quite different. This conception of the relationship between shock and hemorrhage explains the fact, which has been demonstrated so frequently both clinically and experimentally, that a hemorrhage which would be insignificant in the normal state will aggravate or prove fatal in the condition of shock.

Cutler and Scott[60] say:

The immediate effects of severe hemorrhage and shock produce the same syndrome by the same mechanism, viz., a diminution in the circulating blood volume. There is the difference that in shock uncomplicated by hemorrhage the blood is concentrated without loss from the body of the erythrocytes which to a large extent remain in the capillary bed, whereas in case of active hemorrhage the red cells and their contained hemoglobin are lost to the body. This fact is demonstrated by a study of the difference in the recovery of the two types. Thus, after making up the blood volume lost by a large hemorrhage the hemoglobin figure is much lower than after a corresponding degree of shock not caused chiefly by hemorrhage for in the latter instance viable cells are brought back into the circulation.

Rhodes and McKenney[76] make the following distinctions:

If hemorrhage is the major factor, the clinical picture is altered in that: (1) The apathy is replaced by anxious alarm. (2) Instead of the passive immobility of shock there is restlessness and often a marked degree of muscular activity. (3) The shallow rapid respiration is replaced by deeper and more labored breathing, which is terminally aptly described as typical air hunger. (4) The examination of the peripheral blood shows a diminution in red blood cells and hemoglobin as in secondary anemia, thus differing markedly from the findings in shock. There is also a leukocytosis with a relatively high percentage of polymorphonuclear elements, and when the hemorrhage is internal there is a rapid and very marked increase in white blood cells.

Palfrey[28] believes that

the constitutional effects of hemorrhage have one important difference in their course from those of shock. . . . In shock, the symptoms and the fall in the blood-pressure readings occur progressively. In gradual hemorrhage, on the contrary, although the pulse rises progressively, the symptoms tend for a time to be in abeyance, and the blood-pressure tends to maintain itself until at last, quite suddenly, there is a great fall in blood-pressure with serious symptoms of collapse.

Recent Work on Surgical Shock. Concerning recent work on surgical shock, there is in a late issue of the Journal of the American Medical Association[113] the following announcement:

Dr. John Beattie, conservator of the museum of the Royal College of Surgeons, (London), described recent work at the college laboratories on surgical shock. Experimental animals showed three stages. The first fall in blood pressure after trauma was due to loss of fluid. The period of recovery might last thirty minutes and then a gradual fall, terminating in death, took place. The work done in the laboratories pointed to the conclusion that the loss of fluid during the first phase was not the cause of death, which was due to something happening in the second stage, probably associated with impulses passing along the sympathetic nerve. Perhaps these impulses brought about some change in the central nervous system that was irreversible, and in consequence the blood pressure kept on falling until it became so low that death was inevitable. An animal shocked by trauma of the hind limb might die in two and one-half hours. But if the spinal cord was blocked by procaine hydrochloride the blood pressure was maintained and the animal was prevented from passing into a condition of shock. Cross circulation experiments confirmed the conclusion that the lethal factor in shock was abnormal nerve impulses.

Introduction to Consideration of Treatment of Shock. As an introduction to a discussion of the treatment of shock, a statement by McDowall[77] is a propos: "I

propose to . . . emphasize that shock is not a single entity, but may be produced experimentally and clinically from a variety of causes, and that any single remedy used without understanding of the case in hand is bound to give most contrary results."

Measures Directed toward Prevention of Shock. Orr[12] says,

In the civil practice of surgery, serious consideration should be given to the prevention of surgical shock. Much can be done to avoid this complication by properly studying and preparing the patient for operation. It appears obvious that a patient with diseased kidneys, impaired function of the liver, failing circulatory system, cachexia, anemia, toxemia or other conditions which lower the general bodily resistance, might more quickly develop shock than the patient whose vital functions are practically normal. In addition to the careful study of the patient's physical condition, care should be taken not to lower his resistance by ill-advised preoperative surgical preparation. Depleting purges, starvation, dehydration and excessive anxiety should be avoided as much as possible.

In many cases, transfusion, hypodermoclysis of physiologic sodium chloride or infusion of glucose solution before or during an operation will prevent shock. The proper selection of an anesthetic is of utmost importance. Every surgeon should have a good working knowledge of the various types of anesthesia and should make this choice to suit the patient. . . Careful protection of the patient from cold before, during and after operation is essential. Trauma should be minimized always. The judicious use of sedatives to relieve pain and restlessness is not only fair to the patient, but may lessen the shock of anesthesia and operation.

Spinal Anesthesia Advocated in Shock. O'Shaughnessy and Slome[55] advocate spinal anesthesia for shock sufferers. They say in part,

The one therapeutic measure which (we have found) succeeded in raising and maintaining the blood pressure of a traumatized cat whose pressure had sunk to 20 mm. of mercury was the induction of spinal anesthesia. In some cases this measure has been combined with injection of saline into the peritoneum, with apparently beneficial results. It is of course possible that some better method of controlling nociceptive impulses may be devised —the injection of a local anesthetic into the traumatized area suggests itself as one possibility—but we are convinced that control in some form is essential to the successful treatment of the syndrome. Even if we are wrong in the emphasis we lay upon the discharge of nociceptive impulses, the beneficial effect of spinal anesthesia remains a fact which seems worthy of note and further investigation.

Anesthesia and Shock Prevention. Another note regarding anesthesia in shock previously quoted from an article by Jones[39] is repeated here: "local nerve block with a cocaine derivative (nupercain) lessens damage to the central nervous system, allows relaxation with minimal anesthesia, and prolonged freedom from pain in the postoperative period."

Crile's[15] testimony is that "We have found that the acute exhaustion (shock) of surgical operations may be minimized or prevented by blocking the field of operation with local anesthetics, or by preventing the response of the brain-cells to the stimulus of traumatic impulses by nitrous oxide anesthesia."

Cutler and Scott[60] declare that

If the blood volume can be maintained in the early stages before the capillary permeability has been irreparably depressed, the serious phases of shock and hemorrhage never appear and the circulatory efficiency is kept above the critical level. This makes it clear how important it is to be able to estimate the available volume of circulating blood.

These authors say also concerning prevention of shock:

Postoperative hemorrhage and shock are prevented (1) by operating upon patients only when they are in a condition at least as regards their circulatory mechanism, which we designate as "good risk," i.e. their heart action, their vasomotor tone and their blood volume are as near normal as possible; and (2) by utilizing careful surgical technique. This means perfect hemostasis during the operation, care-

ful tying of ligatures, gentleness in handling tissues, and asepsis.

The Dangers of Persisting Low Blood Pressure. Under caption of "Principles underlying proper treatment," Rhodes and McKenney[76] say,

. . . If the low blood pressure with its reduced volume flow is sufficiently persistent, permanent cellular changes are brought about in the higher nerve centers, and these ultimately result in paralysis of the vasomotor center. Herein lies the great need for early treatment in shock for once the vasomotor center has become permanently damaged by the anoxemia, vascular tone is lost. When this loss of vascular tone exists, our efforts to combat shock, particularly those endeavoring to restore fluids, will be fruitless. The problem accordingly resolves itself into an attempt to interrupt the chain of events which leads to these typical vicious circle reactions.

Wallace,[78] who had an extensive experience with shocked patients at the battle front, states that "all the established methods of treatment were used, but warmth combined with rest greatly outstripped all others in favour."

Treatment of Shock More or Less Independent of the Cause. Atchley[45] says,

The treatment of shock is more or less independent of its cause. Whether it is due to trauma, toxemia, hemorrhage or anhydremia, the physiologic problem is the same; namely, a disproportion between the blood volume and vascular bed. On one hand there is primarily decreased blood volume from hemorrhage or fluid loss; on the other increased vascular bed from capillary dilatation. The need for immediate measures to increase blood volume is common to all types.

A point of view opposite from that quoted is found in an article by Andrews.[79] He says, "The treatment for the various sorts (of surgical shock) is quite specific and if applied to the wrong kind, may be definitely detrimental."

Shock a Safety Reaction of Nature. In considering the treatment of burns Clark and Cruickshank[80] make an interesting observation: "Shock in itself may be Nature's effort to deaden pain and tide the patient over the initial phase of his injury, and its treatment should be conservative. Loss of fluid should be prevented as far as possible and replaced by the most suitable methods."

Principles Underlying Selection of Proper Drugs for Treatment of Shock. Says Johnson,[81] of Chicago,

The method of treatment of acute surgical shock or of shock due to hemorrhage when the predominating symptoms are low blood pressure, weak and thready pulse, and depression of the central nervous system has always been unsatisfactory as far as the use of drugs is concerned.

As a rule, in this type of shock a vicious circle is established in which depression of the blood pressure causes further depression of the central nervous system and vice versa. In some instances it appears as if the central nervous system depression is the predominant factor which leads to loss of vasomotor tone with a lowering of blood pressure. In other cases the low blood pressure seems to be the essential factor with consequent central nervous system depression.

In the choice of a drug designed to overcome this vicious circle, several factors ought to be considered, the most important of which are as follows:

1. The drug should be non-toxic with a high margin of safety; i.e., a drug that will not cause a toxic reaction in already depressed circulatory and nervous systems.

2. It should have a pronounced hemodynamic action as well as a marked stimulant action on the central nervous system.

3. These actions of the drug should be of a lasting character in order that the various body systems may regain the physiologic state lost by the previous inadequate circulation of blood through them. If the drug has a lasting action, time will be available for the institution of other supportive measures.

This author[81] states that "ephedrine seems to exhibit these characteristics."

Use of Coramine in Shock. Wood,[82] reporting on the efficacy of coramine, says,

1. Coramine is a definite stimulant to respiration and circulation depressed by avertin, novol, or in surgical shock.

2. The toxicity of coramine is low; so that large and repeated doses may be employed if the necessity exists. This is well illustrated in one of our cases, where 28 c.c. were given in a period of sixteen hours with apparently excellent results. Further, that in this series there was no evidence of depression after the immediate stimulation . . .

Use of Morphine in Treatment of Shock. Orr[12] says, "Undoubtedly morphine should be given in many cases of shock. . . . Crile and Lower have recommended giving morphine until the respiration is reduced to 12 per minute." According to Solis-Cohen,[83] "In conditions of surgical shock, morphine is sometimes the best agent that can be employed. In other cases, strychnine or epinephrine may be preferable."

In Marshall's opinion, "the severely wounded do not make good recoveries from operation if deeply morphinized."[12]

Orr[12] quotes Cannon's report of experimental observations

made by Cattell at Dijon upon the relationship between the use of morphine and the change in alkali reserve. The results show that morphine prevents the fall in alkali of the blood and that, in cases of low blood pressure with an already low carbon dioxide combining power, there is a rise in alkali approaching normal. The explanation for this effect of morphine is not clear, but it is probably due to a reduction of bodily activity, with a resulting decrease in tissue demand for oxygen.

Henderson[84] points out also that "one of the principal effects of morphine is to decrease and in sufficiently large dosage, finally almost to abolish the sensitivity of the neurorespiratory system to its normal stimulus, carbon dioxide."

Regarding the use of adrenalin in shock, MacFee and Baldridge[65] say,

Adrenalin to raise blood pressure is to be condemned. Acting as it does upon the arterioles, its effect is to raise the blood pressure in the arterial tree. But, as pointed out by Cannon, this does not improve the volume flow in the capillaries. " . . . Merely a higher arterial pressure is not the desideratum in the treatment of shock, but a higher pressure which provides an increased nutritive flow through the capillaries all over the body."

These authors[85] declare that "cardiac stimulants, in the absence of organic cardiac disease, are generally uncalled for."

Adrenalin Insufficiency (?) in Shock. Swingle, Pfiffner, et al.[85] express the suggestion that "the signs and symptoms of adrenalin insufficiency, and of traumatic or secondary shock are possibly due to one and the same thing, i.e. failure of the blood volume and blood-diluting regulator mechanism, the adrenal cortex."

The Relationship of Adrenal Cortex Hormone to Shock. There has been considerable criticism and discussion of Swingle's work and publications. Freeman[86] believes

The investigators (under discussion) have clearly demonstrated that the cortico-adrenal hormone is a specific therapeutic agent for the shock which is associated with absence of the adrenals, but they have not reported experiments in which the effect of cortin has been assayed in traumatic shock. . . . Although the analogy between deficiency of cortico-adrenal hormone and traumatic shock is a close one, no convincing evidence has been presented that the two conditions have a common etiology or that the cortical hormone is of benefit in the treatment of shock.

Britton and Silvette[87] say,

Our own observations lead us to conclude that changes in blood pressure and in the amount of circulating blood are indirect and illustrative only of the general effects of adrenalectomy throughout the body. . . . Our data compel adherence to our first proposed theory of the adrenal cortex—that of the regulation, in cooperation with other tissues or secretions, of carbohydrate metabolism in the organism.

Functioning of Adrenal Cortex as Related to Shock. Parkins and his co-workers have done considerable experimental work on the subject of results of adrenalectomy in relation to the shocked state of animals. Their experimentation leads Swingle and Parkins[88] to report in part that "The data

indicate that the unoperated dog has sufficient reserve hormone in his intact functioning adrenal cortical tissue to prevent shock following hemorrhage and trauma. . . . The adrenalectomized animal enjoying good health but lacking such reserve hormone, succumbs to shock as a result of trivial injury."

Shock Causes Marked Reduction in Functional Efficiency of Adrenal Cortex. Experimental data is presented by Donahue and Parkins[89] indicating

> that following trauma and the onset of secondary shock the adrenal cortex is subjected to severe functional strains leading to a marked depletion of lipoid, gross hemorrhages into the gland and vacuolization of cells. These changes are indicative of marked reduction in the functional efficiency of the cortex in this syndrome (i.e. shock).

Parkins et al.[90] have also reported evidence that "The adrenalectomized dog although in equally good physiological condition is far more susceptible to the decrease in serum sodium and chloride and associated disturbance in fluid balance following intraperitoneal injections of isotonic glucose than is the animal with intact adrenals."

Use of Stimulant Drugs in Shock during the Great War. Cannon[13] says with regard to the use of drugs in treatment of shock: "In British and American services the use of stimulant drugs, such as strychnin, and also vasoconstrictor drugs, such as pituitrin and adrenalin, practically disappeared during the course of the recent War." The results of such experience as that on which Cannon bases his statements must always take precedence over conclusions such as those expressed by Harrower,[91] e.g., who says: "For years it has been known that adrenalin offers a life-saving service in shock and heart failure. The best method of administering adrenalin in cases of ordinary shock is by intravenous infusion of high dilutions in saline solution."

Epinephrine and Pituitary Solutions in Treatment of Shock. Summarizing their recent experimental work with pituitary and with epinephrin solutions, Blalock et al.[92] say:

> Experiments were performed in which a decline in blood pressure was produced by the introduction of histamine, of unpurified acacia and of incompatible blood, and salt solution was injected continuously intravenously. The introduction of solution of pituitary or of epinephrine in the amounts used in these experiments did not prevent the loss of protein from the circulation.

Friedlander,[34] also, concludes that

> Epinephrin and pituitary extract are of *no* value in the treatment of secondary shock. . . . And Cannon adds that the desideratum in shock is a higher pressure which provides an increased nutritive flow through the capillaries all over the body. This cannot be brought about by any medication, but only by such measures as will definitely increase the blood volume and thus help the volume flow through the capillaries.

Atchley and Loeb[11] believe that

> The use of vasoconstrictors, such as epinephrine, is not helpful and may, indeed, be dangerous. From a physiological standpoint they are contraindicated because the blood vessels which they affect are already constricted to the disadvantage of the capillary circulation, as has been shown by studies of both the skin and visceral arterioles.

Parathormone in Treatment of Shock. McDonagh[40] proposes that parathormone be used, declaring that "Parathormone is a conductor, it causes dispersion of hydrated particles, and is of equal value in preventing histamine from producing shock, insulin from producing convulsions as it is in preventing guanidine, etc., from producing tetany."

Palfrey[28] says that of the stimulant drugs, "caffein sodio-benzoate, grs. 2–3, given intravenously, is the one most generally believed in." He thinks "epinephrin (1:100), minims xv, added to normal salt solution and given intravenously is of value."

Caffein and Digitalis in Treatment of Shock. Rhodes and McKenney[76] declare

that "Caffein in large doses is used almost routinely as the proper stimulant for the patient in the shock state. Certain observers recommend also various cardiac stimulants of the digitalis group for these patients. The value of both caffein and digitalis is probably over-estimated, as apparently there is no intrinsic myocardial fault demonstrable."

Wangensteen[21] says "Blalock found that digitalis in shock lowered the minute cardiac output, and he concluded that its use in shock is actually harmful. It is to be remembered that the contractile power of the heart is normal in shock and that it is not in need of stimulation."

The Use of Drugs in Treatment of Surgical Shock. Wangensteen says elsewhere,[93] with regard to use of drugs in treatment of surgical shock,

> In the fall of blood pressure accompanying spinal anesthesia and that occurring after sudden removal of large quantities of fluid from the body cavities, the administration of vasospastic agents is urgently indicated, for in these conditions there is an actual lowering of the tone of the vessels. In these conditions too, the Trendelenburg posture has its greatest value.

Later in this same article the author says, "Even though patients in shock suffer more from depletion of blood volume than from want of oxygen carriers, the most effectual means of restoring a diminished blood volume is transfusion of blood."

The conclusions of Cutler and Scott[60] regarding treatment of shock may represent the general consensus of opinion: "Other than transfusion and the administration of fluid, heat and morphia will do more to restore vasomotor tone than all the other procedures advocated."

"Of all methods of intravenous treatment in shock," says Orr,[12] "blood transfusion most nearly approaches the ideal. It will raise the blood pressure, increase the blood volume and, in addition, supply oxygen-carrying corpuscles."

Fluids should be introduced into the body by whatever means is best suited to the particular case. Brandson and Hillsman[94] say,

> The use of fluids is invariably directed towards maintaining the acid-base balance, electronic concentration and blood volume. . . .
>
> To resist the various disturbances to which the organism is constantly subjected these processes have formed a defensive alliance. A disturbance in any one is resisted as far as possible by the specific process involved. When this process is unable to further cope with the situation the other two are called upon for aid, until by mutual assistance the biochemical balance is again established.

Saline Solution in Treatment of Shock. These authors[94] say further:

> In saline we have a solution that will restore very promptly in all surgical conditions the acid-base balance, electronic concentration and the blood volume. The chief base of the blood is sodium; the chief acid is chlorine; the great bulk of the blood volume is water; and the electronic concentration is chiefly maintained by retention, excretion and diffusion of sodium, chlorine, and water. Give to the organism a sufficient quantity of isotonic saline solution and it will by selective action retain sodium and discard chlorine in the presence of an acidosis, retain chlorine and discard sodium in the presence of an alkalosis, and utilize the sodium, chlorine and water to distribute between blood stream, tissue space and body cell, to establish electronic balance and restore blood volume. In order to correct all three processes simultaneously the saline must be isotonic, as saline in any other concentration will affect the electronic concentration and force readjustment of the other two processes.

Hypertonic Solutions. Padgett and Orr[95] say that "hypertonic solution of crystalloids have not been used with complete success in the past in the treatment of well developed shock. Recently, evidence is accumulating that intravenous therapy with hypertonic solutions of crystalloids, unless introduced with the greatest care, may be actually harmful."

Cutler and Scott[60] state that

> It has been shown that the administration of large amounts of fluids (by rectum, by

mouth, and subcutaneously or intravenously) washes out into the general circulation cells which presumably are stored against such an emergency. . . . The spleen may be such a reservoir. The investigations of Robertson and Bock seem to indicate that even immature forms of red cells, possibly from the bone-marrow, are freed into the circulation following the administration of large amounts of fluid. Such cells, of course, have a greater permanency of value than transfused cells, and in all such cases a high fluid intake is essential.

Temporary Effect of NaCl or Ringer's Solution. "Bayliss (Intravenous Injection in Wound Shock, London, 1918) has proved quite conclusively," says Orr,[12]

that the injection of physiologic sodium chloride or Ringer's solution has only a temporary effect in the treatment of severe shock. A rise in the blood pressure follows the injection, but within an hour it usually returns to its previous level. The solution literally leaks out of the blood stream into the tissues. Hypertonic sodium chloride solution raises the blood pressure, but its effect is usually transitory. The high osmotic pressure of the hypertonic solution probably draws additional fluid into the blood stream, but this again passes out through the capillaries as soon as dilution takes place.

Orr[12] quotes Cannon, Fraser, and Cowell as recommending use of 4 per cent sodium bicarbonate solution in treatment of shock, stating that "before or during operation it raises the blood pressure during the critical period. This solution also increases the alkali reserve."

Shelton[86] recommends the use of "a pure 5 per cent dextrose solution in Ringer's solution (which) may be given intravenously continuously for days . . . Usually an intravenous cannula is employed, but for a period of a few hours the intravenous hypodermic needle is often satisfactory. In cases of shock this method is of great value."

Glucose-insulin Treatment in Shock. Wade,[97] in 1929, wrote in summary,

. . . (2) Glucose intravenously with insulin subcutaneously in the treatment of shock gives results which, in this series, seems more satisfactory than those obtained in cases treated by saline or glucose alone. (3) Cases of traumatic shock respond most readily to this treatment. (4) Cases of postoperative shock treated in this manner show marked improvement. (5) The optional dosage is 1000 c.c. of a 5 per cent or 10 per cent solution of glucose with one unit (U20) of insulin to three grams of glucose. Beneficial results are usually apparent after 800 c.c. of fluid have been injected.

The conclusion in regard to the glucose-insulin treatment reached by Padgett and Orr,[95] however, is that "from the experimental standpoint the glucose-insulin treatment of shock recently advocated by Fisher seems to be no more beneficial then treatment with a hypertonic solution of glucose or sodium chloride."

More recent is the opinion expressed in an editorial note[112] published by the Journal of the American Medical Association in response to a query submitted by a Kentucky physician with regard to the use of dextrose intravenously in the treatment of shock:

. . . Aside from supplying water and sodium chloride—the latter is important and should be provided in the solution—during a state when there is a drain on the system without chance for replenishment through the ordinary channels, the dextrose may antagonize starvation acidosis, and that is all the good dextrose-saline solution can do in shock. As this is therefore merely a matter of replacement of nutritional essentials, it has no place in the early treatment of shock. . . . When blood has been lost, it may be of detriment rather than of advantage, as it is likely to lower still further the percentage of corpuscles in the circulating blood. . . . When there has been a great loss of salt from the system, as by excessive emesis, hypertonic (10 per cent) sodium chloride solution may be the most important emergency remedy. Hypertonic dextrose solution should be avoided, as it causes crenation and possible destruction of blood corpuscles when none can be spared; and it will still further increase the existing state of hypohydration of the tissues. . . . One should therefore employ drip phleboclysis,

using equal parts of 5 per cent dextrose and 0.9 per cent sodium chloride solution, as the first offering of diet to a patient who is emerging from shock and who is presumably or actually in such a condition that he is not likely to benefit from the ingestion of fluid by mouth. Excepting in a case of diabetes mellitus, insulin should be employed only when sugar appears in the urine of a patient who is given dextrose phleboclysis and then only in guarded dosage to make hypoglycemia impossible.

As has been noted in the foregoing, Cannon[13] found that the blood sugar in shock is above normal, and that it is not related to the decreased alkali reserve. Acetone bodies were absent from the urine . . . "It seems probable that the beneficial effects of glucose are due largely to the water introduced with it." This statement is made by MacFee and Baldridge.[65]

Glucose and Histamine Shock. Mention should be made of an abstract of an article by Djuricic[98] on the subject of "glucose and histamine shock" which says, "Contrary to what happens in anaphylactic shock, glucose exerts no protective action in histamine shock. This is another fact which is not in accord with the view that the two forms of shock have the same mechanism."

Gum Solution in the Treatment of Shock. "In 1919" says Friedlander,[34]

Erlanger and Gasser* published the results of their experimental studies of shock treated with hypertonic gum solutions. They used solutions containing 25 per cent of gum acacia and 18 per cent of glucose. They found that such a solution acts beneficially in the treatment of shock, (a) by drawing fluid from the tissues to the blood stream, thus aiding in restoring blood volume, (b) by maintaining this increased volume through some property of the acacia, (c) by dilating arterioles through some specific action of the hypertonic crystalloid, (d) by increasing the energy of the heartbeat in the same way, also through the direct action of glucose on the heart muscle, (e) by augmenting metabolism through increase in the supply of glucose to the organism.

* *Ann. Surg.*, 69: 389, 1919.

Randall,[99] reporting on the use of intravenous solution of acacia in the Mayo Clinic in the treatment of shock, makes the following statements:

The total number of patients in all services at Mayo Clinic who received intravenous injections of solution of acacia in 1928 was 118 . . . Acacia apparently fulfils all the requirements for intravenous treatment of shock except furnishing erythrocytes. It has been proved to be innocuous in several series of cases. It increases the volume of plasma and blood and keeps it increased until the normal fluid-regulating mechanism of the body is restored. As preventive of shock I feel that it is often well to give an injection of solution of acacia in cases in which the patient is fatigued or debilitated and in which obstetric operations are to be performed.

Huffman[100] summarizes his findings with acacia and sodium chloride solution as follows:

Gross evidence of toxicity was not seen following the intravenous injection of solution of acacia and sodium chloride in shock. The blood pressure increased, the pulse rate decreased, the respiration deepened and the general condition of the patient improved. The need for transfusion was often obviated. In some cases, increased output of urine was noted. Injurious effects on the kidneys were not seen. Chemical changes in the blood of a harmful nature were not apparent. Physicochemical changes in the blood did not lead to harmful alterations in physiologic processes. Six days is usually required to rid the blood of acacia. Pathologic change resulting from the acacia was not demonstrated at necropsy. The therapeutic usefulness of solution of acacia and sodium chloride is confirmed.

Macleod[75] notes "two precautions necessary to success in using the gum solution, first they must be properly prepared, and second they must not be injected so rapidly that their high viscidity would slow the circulation and so embarrass the heart's action." Erlanger, Gasser, et al.[101,102] early presented several articles reporting favorable experimental results with gum acacia in treatment of shock. Good and Boyer[103]

are among more recent advocates of the use of this solution.

Unfavorable Comment Regarding Intravenous Use of Acacia. Lund, of Boston, in discussing an article by Frazier[104] says,

> I think . . . that the acacia problem is not finished. Acacia stays in the blood a long time. It has been recovered six months or longer after introduction. It is a gummy solution and, according to a pharmacologist, tends to coat and clog the capillaries in the liver. In reasonable amounts, I am sure that it can do nothing but good, but I think that repetitions of doses of acacia, say, 500 c.c. three or four times in twenty-four hours, and three or four times more in the next twenty-four hours, might be bad, and I would like to see animal work done to find what the upper limit of tolerance of acacia in the way of dosage is.

In the consideration of ways and means of administering fluids by the intravenous routes, Wenger[105] says,

> I have found it difficult to give large quantities of fluid intravenously with the usual gravity method. Among other objections, this gravity method is too slow. Because of these inconveniences, the Scannell apparatus for transfusion was used. However, it is cumbersome and somewhat complicated. The apparatus described below is an outgrowth of these difficulties.
>
> *Apparatus for Intravenous Treatment of Shock.* A glass T-tube is inserted between the gravity tube and the needle, which needle should preferably be of the type with a shield to rest on the patient's arm. To the perpendicular of the T-tube, by means of rubber tubing, a 50 c.c. Luer syringe is attached. The other equipment required is the ordinary infusion bottle for transfusion and an artery clamp.
>
> The artery clamp is placed on the tubing just above the T-tube. When the clamp is removed the fluid flows into the vein and into the syringe displacing the plunger. When the syringe is full, the clamp is reapplied; gentle pressure is exerted on the syringe, thus forcing the fluid into the vein. The clamp is removed and the procedure repeated until sufficient fluid has been given. In my cases 500 c.c. are given in a period of from thirty to forty minutes. In no case has there been any ill effect from giving the fluid at this rate.

Wenger[105] declares that

> the advantages of the apparatus lie in its simplicity; in the fact that the treatment can be given at the bedside; that the patient is but slightly disturbed; that a large volume of fluid may be given in a minimum of time; and that the flow into the vein is continuous. Thus there is no clotting in the needle.

Four Dangers Associated with Administration of Solutions Intravenously. In his discussion of an article dealing with the role of intravenous drip in therapy by Hyman and Touroff,[106] Orr points out four major dangers attendant on administering solutions intravenously: (1) the immediate reaction; (2) the possibility of overloading the circulatory tree; (3) the production of edema; (4) the production of pulmonary embolism.

Treatment of Electrical Shock. Brief notice may appropriately be made of the treatment of electrical shock. Pearl's[29] summary and conclusions on this subject include the following:

> . . . (6) The treatment of electrically induced ventricular fibrillation is prompt cardiac massage, preceded if possible by the intraventricular injection of potassium salts followed by calcium salts. The carotid route for administration of these salts may prove sufficient without the use of cardiac massage. If available an appropriate current may be passed through the heart, followed by cardiac massage.

Electrosurgery Technique. It is interesting to note that Schorcher[4] found that "trauma of an electrosurgical procedure produced less shock than that produced by ordinary mechanical means . . . Electrocoagulation destroys the toxicity of the rapidly working toxins because it generates a temperature of 60 to 80 degrees, whereas their activity is inhibited at 59 degrees of heat." The author "believes that he demonstrated in experiments that electrosurgical trauma produces little reaction on the part of the nervous and circulatory systems."

Artificial Respiration in Electrical Shock. "Jaffe . . . joins with other recent stu-

dents," says an editorial in the Current Comment section[107] of the Journal of the American Medical Association under date of June 16, 1928, "in stressing the importance of vigorous attempts at artificial respiration. This may effect resuscitation of a paralyzed respiratory center. The passive movements of the chest should be started as soon as possible and continued for a sufficient time, even for hours."

In discussing application of heat as an important procedure in the therapy of surgical and traumatic shock, it may not be amiss to call attention to the use of diathermy for this purpose. The first paragraph of a paper read at the sixth annual meeting of the American College of Physical Therapy by Portmann,[108] of the Cleveland Clinic follows:

Diathermy in Treatment of Shock. The use of diathermia in the treatment and prevention of surgical shock is based upon certain experiments which were made a number of years ago. Dr. Crile found that cooling of the liver is one of the most important factors in the production of shock in abdominal operations. In a series of animal experiments Nagelschmidt found that the amplitude and volume of respiration and the heart beat are increased when diathermia is applied through the mediastinum.

Description of a Device for Application of Heat in Treatment of Shock. Hitchcock and Reynolds[109] describe an apparatus originally fashioned to expedite cast drying where more or less extensive body casts are required, and later utilized to prevent and to treat cases of shock.

The apparatus consists, essentially, of a cabinet with electrical coils for heating, together with a blower which delivers this warmed air into flexible tubes. These tubes fit, by means of simple coupling devices, into a coiled spring mat which can be laid on a bed or folded around a patient, as any ordinary blanket or mat would be. The coiled springs are covered by fabric, which makes the whole unit into a flexible chamber or a flexible hollow wrapping, through which the heated air circulates, to leave by a vent at its distal end. The principal mat, which envelopes the body and legs, is augmented by an auxiliary mat, which can be fitted over an arm to which a separate pipe can deliver heated air. The amount of heat delivered is subject to simple regulation by means of switches, so that a greater or lesser degree of heat can be obtained, depending on the needs. . . . This apparatus provides "quick, generalized heat" which "is an important factor in recovery from shock."

Carbon Dioxide and Oxygen in Treatment of Shock. Henderson[84] has pointed out recently that "hemorrhage produces its ill effects largely through asphyxia. This is tacitly recognized by surgeons in their preference for transfusion of blood over any mere infusion of a saline or gum solution. The victim of acute exsanguination exhibits air hunger." He recommends treatment with resuscitation methods—the administration of carbon dioxide and oxygen. He says in part:

In shock without hemorrhage there is, however, no loss of red corpuscles from the body, and the blood alkali is merely displaced. Both corpuscles and alkali may be recalled into use. To effect such recall, both the asphyxia and the acarbia may be combated with inhalation of carbon dioxide and oxygen.

One of the oldest and also the latest of many conceptions of the underlying cause of the depression of the circulation in shock and in illness is that the tonus of all the muscles of the body, both skeletal and visceral, is depressed, and that the blood stagnates in the atonic tissues. Stimulation of respiration with carbon dioxide increases the effective differences of pressure between the tissues of the body and the thorax and thus promotes the venous return to the heart.[84]

Support for the Theory of the Value of Carbon Dioxide in Treatment of Shock. Support for Henderson's claims are found in reports of work by other investigators. McDowall[37] says,

The tone of the blood-vessels is kept up by the vasomotor center, the activity of which Dale and Evans have shown to be dependent on the carbon-dioxide content of the blood. It will be clear, then, that any condition which separates the centre from the vessels or which causes the carbon dioxide to be reduced must

result in a fall in blood pressure from not only an increased capacity of the circulation but also a loss of the peripheral resistance of the arteries.

War Experience with Carbon Dioxide in Treatment of Shock. War experience, also, serves to establish validity for Henderson's theory of treatment for shock. Porter[44] says in the summary of his lectures on shock: "#13. Increased action of the thoracic pump, brought about by the inhalation of carbon dioxide liberally mixed with pure air, will raise the blood pressure from 15–20 millimetres in normal and wounded men. When the blood pressure is near the critical level this procedure is of advantage." Previously this author[44] had made a point of the fact that "A few mms. above the critical level, recovery will usually occur spontaneously; a few mms. below, death will follow unless skilled aid be at hand."

Keen,[110] quoting Porter largely, says:

> All physiologists know that the pumping action of the diaphragm is an important aid in the movement of blood from the abdomen into the chest. At the height of a strong inspiration the venous pressure in the chest may be more than 40 mms. lower than the venous pressure in the abdomen. I produced strong respiratory movements of the diaphragm by allowing the animal to breathe an atmosphere rich in carbon dioxide. The *diastolic* arterial pressure (which should always be the guide; the systolic pressure should not be used as the standard) was thereby increased 15 and even 30 mms.
>
> In June, 1917, I successfully applied this new method to the treatment of wounded soldiers. In cases almost pulseless, cases in which all other means of raising the blood pressure had failed, the carbon dioxide respiration strengthened the pulse and raised the diastolic blood pressure 10 mms. This rise is of great value when the pressure is at the critical level.

Postmortem Findings in Shock. Moon and Crawford[46] give the following postmortem changes as characteristic of shock: "widespread dilatation of capillaries and venules, with edema and capillary hemorrhages. These were most marked in the organs of respiration."

Wakeley and Buxton[19] say that

> Postmortem, very little of importance is found in shock, the only reliable findings being in the central nervous system, where . . . chromatolysis occurs, commencing on the afferent side, and extending to the important vital nuclei. This is regarded as due to the nociceptive stimuli received wearing out the afferent nuclei first, extension being a secondary matter. Chromatolysis in the cerebellum has been shown to be very constant. Hence we see how profound is the benefit of sleep.

Moon[16] says that "Shock produces characteristic changes demonstrable by postmortem examination. These consist of marked capillary and venous congestion of the viscera, edema of lungs and mucosae, petechial hemorrhages in serous and mucous surfaces, and effusion of fluid into serous cavities."

SUMMARY

A very little reflection on the part of the reader will lead to the realization that, long as this review is, it represents in its present extent a virtual summarization of the available literature of the subject. It is probably not possible to condense further, i.e., to summarize, and at the same time to keep the review representative of the actual literature.

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RUPTURE OF BLADDER AND URETHRA*

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IN 9900 successive urologic admissions to the Squier Clinic, there were 20 cases of bladder injury and 27 renal injuries. In general, rupture of the bladder is an infrequent injury. The incidence has been reported to have occurred once in 5550 times, in over 300,000 surgical admissions to Bellevue Hospital. There is an apparent increase of bladder ruptures as evidenced in the recent literature.

Injuries to the bladder are of two types. The first is, rupture, due to sudden increased internal hydrostatic pressure and the second, puncture or perforation, due to the passage of some foreign object or adjacent bone.

Rupture of the bladder is the more frequent of the major injuries to the bladder. It occurs more often in males because of the greater hazard of exposure to traumatism. The highest proportion is that of Campbell with 3 females in 55 collected cases. In the Squier Clinic there were 3 females in 13 cases of rupture. The injury is most frequently seen in adult life but again from the literature, the proportion in childhood is increasing, due to the marked number of automobile accidents.

Rupture or bursting of the bladder can only occur when the bladder contains fluid. The more fluid it contains the more easily is it ruptured. The empty bladder is cushioned within the pelvis and well protected, therefore it is infrequently injured. The full bladder is mostly an abdominal viscus, belongs by attachments to the anterior wall and is exposed with and suffers from its traumatisms. The most common cause of rupture is some external violence as blows, crushes or falls. This is especially so in those conditions that allow the bladder to become overdistended. Foremost among these is alcoholism. The partial anesthesia of alcohol allows the bladder to overfill, especially with the diuretic effect of alcohol, and again the partial anesthesia prevents the usual anticipation of trauma, so that there is no protective muscle contraction to protect the bladder. Other conditions that permit the normal bladder to overfill are psychopathic states, anesthesia, intensive mental occupation and social situations which prevent relief, not uncommon in automobile transportation. Pathological conditions that weaken the bladder wall, as atony, obstruction, tumors, or diverticuli and so forth, tend to make such bladders more prone to injury in minor traumatisms.

Spontaneous rupture has been reported. Rupture has occurred from muscular effort alone. It is thought that in these types, there must be some local weakness that makes these bladders more susceptible to trauma.

Rupture is frequently associated with pelvic or other fractures. Bartels found 65 bladder ruptures in 169 fracture cases. Campbell reported 25 cases of bladder injury in 166 cases of fracture of the pelvis. De Tarnowsky reported that only 5 per cent of his bladder ruptures were caused by or accompanied fractured pelves. Boorstein found 6 ruptured bladders in 100 pelvic fractures at Fordham Hospital. In the 20 cases of bladder injury at the Squier Clinic there were 5 fractured pelves. In respect to automobile accidents Bacon and LeCount, in a study of 383 automobile deaths, found 42 cases of fractured pelvis

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but only 3 with bladder ruptures. They estimate 0.7 per cent of automobile accidents are accompanied by bladder rupture.

Rupture may occur from sudden increased pressure from within the bladder. Wilhelm, Fillis and others have reported cases occurring at the time of cystoscopy. Ruptures have been reported as having occurred during litholopaxy. More recently cases of rupture have been reported by the explosion of gas generated by prostatic electrical revisions and there undoubtedly are more unreported, following the enthusiastic spread of this procedure. Self catheterization and simple bladder irrigations have been blamed for bladder rupture. Parmalee, Pridham and others have reported bladder rupture during pregnancy and Keenan, Roxas and Rustia have reported the accident during puerperium. Perforation or rupture of the bladder from extravesical pathological processes in organs in the vicinity have been reported, among which, was the unusual accident reported by Fillis, of a gravid uterus rupturing into the bladder.

MECHANISM

The bladder ruptures like any fluid sac subject to sudden pressure in one place or as any fluid sac that is moving and suddenly arrested by contact. There is an instantaneous shortening of the poles of the line of contact. The fluid, incompressible, immediately expands the circumference equatorially to the poles and the rupture occurs at its weakest point or most expansile portion. The usual trauma is to the anterior abdominal wall. The poles would be anteroposterior, the equatorial circumference vertical and the unsupported expansile portion, the dome or superior-posterior surface, and the rupture would occur at this point, intraperitoneally. This is the more frequent lesion. In falls on the buttocks, the full bladder acting like a bag will flatten vertically in its line of contact, and expanding its equatorial circumference, can only do so laterally or posteriorly, either intra- or extraperitoneally, where it is unsupported and will rupture at this area. Such ruptures or tears are widest at the equator and run towards the poles. It is possible, with a fractured pelvis, to have either a ruptured bladder or a perforated bladder from the fractured bone and diagnostically, before operation, such determination may be impossible.

In the cases of perforating wounds due to fracture of the pelvis, the pelvis is crushed, the rami are fractured and the pelvis again rolled between two opposing forces and the already fractured rami are forced into the pelvic tissues and through the bladder wall. Such forces may more easily injure a distended bladder, but do also injure a bladder comparatively empty. Crushes separating the pubis not infrequently tear the tissues down through the sphincteric portion of the bladder.

SYMPTOMS

The symptoms are first those of shock. Campbell reported that one-fourth of the cases at Bellevue Hospital were in shock on admission. The shock may be slight or severe, and with the shock frequently occur the signs suggestive of hemorrhage. Local pain and tenderness in the lower abdomen are usually present. In alcoholics or in psychopathic cases little complaint may be given concerning their local reactions. Increase of local tenderness progresses with the symptoms of infection and with peritonitis, if intraperitoneal. The literature has many extraordinary cases where these signs have been overlooked. There is frequently a marked desire to void, with inability to produce urine or at most a few drops of blood. Gastrointestinal symptoms may be absent until peritonitis develops. Frequently the tenderness present with a fractured pelvis may mask or cause to be unrecognized the true abdominal tenderness. It has not been unusual to find the attention focused upon some associated injury or fracture and the abdominal symptoms neglected until all are predominant.

The symptoms may be all those of a fractured pelvis with pain, inability to walk or to use the lower limbs without pain, and the desire to void may be present only to a slight degree.

Fig. 1. Intraperitoneal and extraperitonel diffusion of iodide solution in a bladder rupture associated with a fracture of the left side of the pubic arch in a thirty-four year old female.

DIAGNOSIS

In any case with a clear history of injury and with the sensation of a sudden "giving way" in the abdomen, with inability to void, there is a strong possibility of rupture of the bladder. Frequently such a history is not obtainable. Examination shows abdominal distention in over 60 per cent of the cases. Free fluid in the abdomen is demonstrable in 30 per cent of the cases. Hematoma may be present somewhere in the region of the pelvis. The most commonly employed procedure for diagnosis has been catheterization. No urine or a small amount of bloody urine may be obtained. Deduction may be difficult, if the catheter eye is plugged with blood which frequently may be traumatic from the catheterization. Occasionally a catheter penetrating through an intraperitoneal rupture will drain a large amount of bloody fluid. Instillation of a measured amount of fluid with inability to recover the entire amount may suggest a rupture. A recently seen rupture occurred, in which the exact amount of fluid was recovered several times.

Cystoscopy has been used to demonstrate rupture of the bladder. Definite determination often has been impossible on account of bleeding or the inability to keep the bladder sufficiently well distended to visualize the whole. Both catheterization, injection and cystoscopy add additional trauma and increase the incidence of infection. If used, these should immediately precede exploration.

Roentgenograms of iodide solutions injected into the bladder will give a shadow of the penetration of the iodide outside the bladder. Vaughn and Rudnick introduced air in 50 to 200 c.c. amounts, followed by *x*-ray pictures for the same purpose. The air has been shown, in cases, to ascend under the diaphragm, definitely proving intraperitoneal rupture, but may confuse a possible intestinal rupture, unless its absence has been previously confirmed. Such injections occupy the same position

as cystoscopy. They increase the incidence of and add to the spread of infection.

Of most value have been the cystograms obtained from injected intravenous pyelographic solutions. They lessen the most serious risk, that of infection. It is not always possible to delay operative intervention in the more serious injuries, so that a secretory cystogram may be obtained. Such injured should be given a transfusion followed by operative exploration which should reveal the injury. However, the larger number of cases allows such procedure and in these, it demonstrates the urinary extravasation as well as traumatic lesions of the kidney and ureter.

A preliminary *x*-ray study of the chest and abdomen will disclose associated fractures or the possibility of other visceral injuries. When there is considered the marked frequency of a bladder injury to be associated with a fractured pelvis all fractures of the pelvis should have intravenous urographic study as early as possible. In fractures of the pelvis, however, the passage of clear, normal voided urine in any quantity, has been considered as ruling out the possibility of bladder injury.

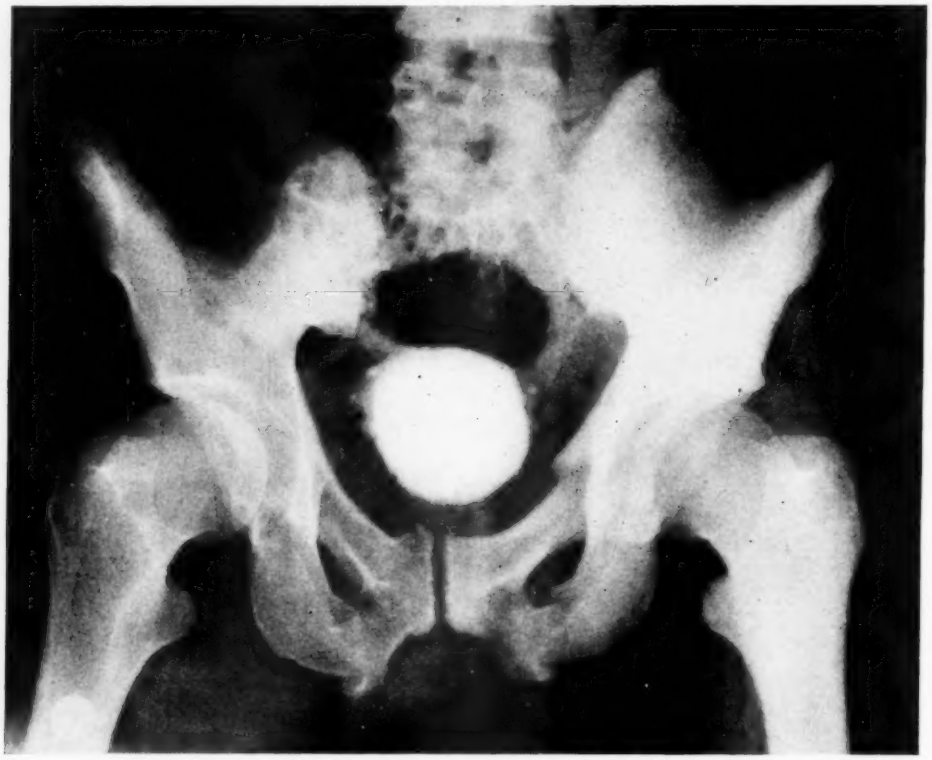

Fig. 2. A secretory cystogram showing a fracture of the four rami of the pubis with intact bladder with small cellules and the bladder displaced upward by pelvic tissue diffuse hematoma. Injury to left kidney with fractures treated by transfusions and rest and care of fractures. Recovery followed without operation.

Early blood examinations are the index of the degree of hemorrhage; later examinations are an index of the severity of the infection. Other laboratory examinations, except the demonstration of blood in the urine have been of no special significance.

TREATMENT

Having established the probability of the diagnosis, immediate operation is essential. If shock is present, the condition has been best improved by transfusion, associated with heat and stimulants. There is no special choice of anesthesia but low blood pressure may contraindicate spinal. If in an alcoholic, a preliminary gastric lavage may prevent a later insufflation pneumonia.

Intraperitoneal exploration is necessary in most cases. If intraperitoneal involvement is present, the fluid is aspirated. The vesical tear is located after exploration has disclosed no other visceral injuries. The tear is closed with special attention to peritonealizing the closure. After complete aspiration of all the intraperitoneal fluid, the peritoneum is closed, if the case is early. If so, then ample aspiration suprapubic drainage of the bladder with a wide tube is provided. This drainage, we find keeps the bladder dry and prevents dilatation and leakage.

Associated injuries to other abdominal viscera may occur. The more frequently seen have been to the intestine. Next in order are injuries to the spleen, the kidneys and the liver. Correction of these injuries, if possible, is performed at the time of exploration. With the presence of blood in the urine and a ruptured bladder, there is still a possibility of renal injury. To establish this particular point the intravenous secretory pyelographic studies may be of great importance.

Perivesical hematoma or extravasations are widely drained through the suprapubic prevesical opening. If the case is seen late and evidence of local peritonitis is present, drainage of the intraperitoneal area suprapubically should be attempted with a walling off of the peritoneal cavity with the omentum.

Fig. 3. A suspected bladder injury with abdominal blow and left flank stab wound. A secretory cystogram shows an intact bladder but a huge blood clot that caused urinary retention. The bleeding was from the left kidney.

If the injury and extravasation are extraperitoneal, the peritoneum, if opened, is rapidly closed and then the procedure is modified accordingly, to the area involved. Penetrating wounds from fractured bones are frequently small, but require suture, because they bleed annoyingly. Occasionally the bone remains penetrating into the bladder and removal may be difficult. It has been necessary to remove the projections or spicules before the bladder can be freed from empalement. It may be wise in such conditions to endeavor to close such bladders completely and drain with an

indwelling catheter. From past experiences, urine draining across fractured, exposed bone, has been followed by a very serious osteomyelitis and marked absorption of the calcium of the bone.

Extensive lacerations of the bladder and pelvic tissue may occur in which complete repair may be unwise on account of the precarious condition of the patient. It is safer to control the hemorrhage, provide wide drainage and restore vitality with transfusions and so forth.

Injuries involving the floor of the bladder and with extravasation into the deep pelvis, may be drained perineally as well as suprapubically. Some writers, Berry for instance, preferred perineal section and drainage with his cases of ruptured bladder. In the series of cases reported by Campbell, external urethrotomy with drainage, was attempted four times with no recoveries.

SURGICAL PATHOLOGY

The urinary extravasation may extend into the subcutaneous tissues or cause a marked edema of those tissues and the abdominal muscles. Extraperitoneal extravasations cause early, marked reddish edema of the perivesicular tissues which later change into grayish or grayish red tissues that do not reflect light. Localized collections of watery abscesses may occur. Marked perivesicular hemorrhage may obscure the bladder musculature so that recognition of the bladder may be difficult. Crepitation is not uncommonly present. The infection may or may not be from the gas bacillus of Welch but such infections have occurred, especially in the perforating pelvic injuries.

The most frequent site of rupture is on the summit of the posterior surface. It is usually irregular, may be oblique, longitudinal or transverse. The contraction of the bladder may change the apparent shape of the opening. The locations of the sites in Campbell's report were dome 37, lateral wall 4, floor 3, vesical outlet 4 and posterior wall 1.

The perforating injuries from bone are usually on the lateral anterior wall near the vesical outlet and are small. Profuse hematomas are often present because of the vascularity of this area. Similar hematomas occur when the bladder neck has been torn with a pubic separation.

Peritonitis, if present, is more marked in the pelvis and occurs with high frequency as reported in the Bellevue series, having occurred in 23 of 32 intraperitoneal ruptures, due to tardy recognition of the condition.

When the rupture has been associated with previous infection or some previous pathology, the extension of the infection has been marked due to the explosive action of the fluid penetrating throughout the peritoneum. In 3 cases of rupture of diverticuli and in one associated with bladder tumor at the Squier Clinic, a fatal, rapid, diffuse peritonitis occurred in all.

COMPLICATIONS AND SEQUELS

The complications are mainly shock, hemorrhage and infection.

Primary shock and hemorrhage may be rapidly fatal. This is the not infrequent rapid ending in severe pelvic crushing. In some cases in which the shock and hemorrhage have not been severe, an early marked infection develops in the extravasated tissues, with a sepsis and death within several days.

In alcoholics a bronchial or aspiration pneumonia has not been infrequent, especially if vomiting occurred during the administration of the anesthetic.

The association of other injuries, as fractures, lacerations or injuries to other viscera add many complicating factors to the treatment.

Acute osteomyelitis may develop in a fractured pelvis, especially when associated with a urinary sinus. In one case the entire pubic arch was absorbed before the patient died of exhaustion.

Gas bacillus infection may prove rapidly fatal and the extension has progressed as high up as the thoracic area.

Peritonitis has been reported to have occurred in over 50 per cent of the cases.

Postoperative evisceration has occurred in alcoholics and psychopathic cases and in these cases reported hopeless.

Slow healing of the suprapubic sinus is frequent, especially in extraperitoneal ruptures. Some of these cases are due to scar contraction of the injured area, near the vesical neck and require surgical procedures for closure. Residual intrapelvic abscess is not infrequent and is often due to retained slough or insufficiently drained fascial plane areas. Vesical calculi have been reported to have occurred following bladder trauma. Renal infections have been not uncommon with the infection stage and deaths have been reported from subsequent renal deficiency.

MORTALITY

A historical review has shown a definite but gradual lowering of the mortality rate. In a series of cases reported by Besley up to 1907, there was a mortality of 78 per cent. From 1927 to 1929, in 13 cases admitted to the same hospital there was a mortality of 61 per cent. Rouvillers and Ferron, in 1931 tabulated a mortality of 49 per cent in a review from the literature of 278 cases. Before 1894, the mortality in intraperitoneal ruptures was 60.9 per cent, from 1894 to 1921 it was 23.9 per cent. Hagenbruck in 35 cases occurring from 1905 to 1925, reported a mortality of 53 per cent. De Tarnowsky in 1930 had a mortality of 45 per cent in 50 cases. Campbell in a review of all services at Bellevue, found among 55 cases that the mortality was 68 per cent among the intraperitoneal ruptures and 40 per cent among the extraperitoneal. White in 1933 estimated the mortality then as 40 per cent. In 20 cases of bladder rupture and perforation at the Squier Clinic since 1929, the mortality was 30 per cent.

Intraperitoneal ruptures have had the higher death rate. This has been largely due to the failure to diagnose the condition sufficiently early so that operation was not performed in the early period of contamination and before infection was established. The marked seriousness of disseminated contamination in the peritoneal cavity and its prevention by early interference has been so dramatically established in the results established by early recognition and correction in perforating gastric or duodenal ulcer. In bladder rupture similar facts have been well shown by various writers. Negley, in 1927 showed that the mortality in operations performed in the first twelve hours was but 11 per cent, whereas in the second twelve hours it had risen to 22 per cent and by the second day to 43 per cent. Similar tabulations have been reported by Rouvillers and Ferron, and de Tarnowsky. The inescapable conclusion from these facts is that most of the bladder ruptures are seen by those who either are indifferent to the seriousness of the condition, which is a major catastrophe of the abdomen requiring immediate interference, or else they are unaware of the symptoms and signs necessary for the diagnosis.

Extraperitoneal ruptures are considered less dangerous, if uncomplicated. The comparatively less serious extraperitoneal infection, its better localization and with better natural defensive barriers offer a less serious prognosis. Extraperitoneal ruptures in the presence of complications, especially marked hemorrhage and severe crushing injuries to the pelvis, present cases of injury second to none in severity, and with the multiplicity of lesions the mortality rises rapidly.

Most writers in stressing the importance of early diagnosis and operation believe the mortality could be reduced to 20 per cent. The low limit must necessarily be estimated by the degree of general injury in which bladder rupture often plays an important part.

RUPTURE OF THE URETHRA

The urethra may be ruptured by traumatism from within or without. Lack of skill and gentleness in instrumentation is

the usual cause of injuries from within. Less rarely, self-inflicted injuries occur in psychopathic states. The first symptom is continuous bleeding from the urethra, independent of voiding. Control of the bleeding in the milder injuries is accomplished best by pressure upon the corpora spongiosum or perineum, although if accompanied by urinary difficulty an indwelling catheter may be necessary to prevent extravasation. If the bleeding is uncontrolled and extravasation is beginning, as shown by perineal tenderness and swelling, for it is the bulb which is more frequently injured, a perineal section and drainage may be necessary. Rarely, the urethra may be ruptured by injection of solutions. Such ruptures often cause severe phlegmons, which in healing cause marked stricture formation. Among the most severe, have been those cases in which liquid paraffin has been injected for lubrication into the urethra, rupturing it and extravasating through the corpora.

The usual causes of urethral rupture are blows or falls on the perineum. The urethra may be completely or partially severed by a crush against the pubic bone or may be lacerated by the sharp end of a fractured pelvis. The more frequent injury is the straddle; in falling astride a fence, or bar, or saddle horn, or rail, or some narrow object, the full force of the fall being borne directly on the perineum. A similar traumatism is described by O'Conor from a blow to the perineum by a tilting manhole cover.

The tear of the urethra may be transverse, oblique or longitudinal. It may involve any one of the following parts: pendulous, bulbous portion anterior to the triangular ligament and the membroprostatic portion posterior to the triangular ligament. Mainly it is extrapelvic or intrapelvic. The urethra may be completely torn from the bladder. This has occurred with complete, wide separation of the pubic arch. A similar injury occurred in a case admitted to the Squier Clinic in which a complete separation of the urethra from the bladder occurred during a forceps delivery. Recovery followed suprapubic drainage and later urethral anastomosis.

Peacock and Hain collected 200 cases of urethral rupture and found that 8 per cent were due to straddle falls, 12 per cent to perineal blows and 4 per cent to crushes. They found 71 per cent of their cases of traumatic ruptured urethras were associated with pelvic fractures. From their report they concluded that of all cases of fractured pelvis less than 10 per cent have ruptured urethras. Young quotes Baer in estimating that the urethra is wounded in not over 1 or 2 per cent of pelvic fractures.

The first symptoms of rupture of the urethra are pain and urethral hemorrhage. This is followed by hematoma, if the rupture is in front of the triangular ligament, frequently with inability to void. The bleeding is from the meatus and separate from any act of voiding. If the injury is posterior to the triangular ligament, there may be little or no meatal bleeding, but a deep perineal hematoma may be present. Often with intrapelvic injuries the hemorrhage may be all through the perivesical tissues and perineal ecchymosis may develop later.

There is usually not the marked desire to void seen in bladder injuries. Attempts to void, however, are very painful due most likely to extravasation during the act. Marked increase in the tumefaction present may occur immediately following such attempts. The retention of urine is frequently so marked that the distended bladder may be visible or palpable above the pubis.

When extravasation occurs, the course is dependent upon the site of the rupture. Extravasation superficial to the triangular ligament will be limited by the attachment of Colles's fascia and will extend into the perineum, the scrotum, and upwards to the inguinal regions and abdomen. When the site of the injury is deeper inside the triangular ligament, the extravasation extends about the vesical neck, lateral to the bladder and into the prevesical spaces.

The diagnosis of urethral rupture may be obscured early, in partial ruptures, but as a rule the lesion becomes evident. In the beginning it may be confused with bladder rupture, but later, bladder distention rules out rupture of that organ.

Attempts at urethral catheterization have been widely used as a diagnostic procedure. Such has been condemned by White, Hansen and others, as producing further hemorrhage and introducing infection.

The surgical indications are diversion of urine, prevent or drain infection or extravasation and repair the tear. Hansen recommends observant treatment in the milder cases but for all others an immediate suprapubic cystotomy and later suture of the urethra without a residual catheter. Wheeler believes that all cases should have an immediate suprapubic drainage. He supplements this procedure with retrograde catheterization and then perineal exposure and repair.

In our experience the suprapubic drainage accompanied by continuous aspiration has been most adequate for the urinary diversion. In the presence of urinary extravasation, wide incision and drainage is necessary. The repair of the urethra is done at times with difficulty. Localization of the urethra is best by retrograde catheterization. The interlocking sounds as suggested by G. G. Davis facilitate this maneuver. Early union of the urethra is desired and if the condition of the patient permits should be done at the same sitting. There has been considerable controversy as to whether the urethral repair should be followed by a retention catheter. Some surgeons condemn it because of the constant association with infection. Experience has shown the operative surgical repair is facilitated by the catheter but it should then be removed and the aspiration suprapubic suction drainage used to divert the urine until healing has taken place. Repair in a perineum may not be possible when extravasation and infection are marked. Such cases are best treated by wide incision, extirpation of as much necrotic tissue as seems expedient, and as soon as healing has commenced a repair of the severed urethra attempted. These cases are frequently followed by severe intractable urethral strictures necessitating long periods of urethral dilatation. Plastic repair may be necessary for urethral defects or for undilatable strictures. However, these are more successful if undertaken after the inflammatory reaction has subsided.

Impotence is not infrequent after urethral injuries and plays an important place in those cases under settlement for compensation disabilities. The impotence has been described by Peacock and Hain to be due to lesions of the sympathetic fibers with the urethra, injured at the site of rupture.

The prognosis of urethral rupture is better than vesical rupture as far as life recovery is concerned. Hansen reports a mortality of 29 per cent in 135 cases, but if the hopelessly injured with other involved organs were excluded the mortality was 14 per cent. The local recovery or perfect anatomical healing is poor. Less than 10 per cent recover without the necessity of dilatations. Most require long and persistent postoperative treatment. Satisfactory functional recovery depends on early diagnosis and prompt and intelligent surgical intervention.

SUMMARY

Cases of bladder rupture are increasing with the increase of automobile accidents. The usual cause of ruptured bladder is external traumatism to an overfilled bladder, not uncommonly seen in alcoholics. An increasing proportion is associated with fractured pelvis. Occasionally the rupture is secondary to medical manipulations. Intraperitoneal ruptures are more frequent than extraperitoneal ones.

Symptoms are shock, abdominal pain and tenderness with marked desire but inability to void.

Diagnosis is best made by intravenous pyelography and cystograms showing the diffused dye outside the bladder. This procedure lessens the danger of infection.

Rupture of the bladder is a major abdominal catastrophe and requires immediate surgical intervention. Closure of the tear with wide drainage and aspiration is recommended.

Complications are shock, hemorrhage and infection.

Mortality has been steadily declining, from 78 per cent up to 1907, to 30 per cent in the tabulation of the cases at the Squier Clinic. This percentage might be still lower if the bladder ruptures could be isolated from the high percentage of general injuries with which they are associated.

The urethra may be ruptured either from within or without. Rupture from without is the more usual and is generally due to a fall on the perineum. It frequently occurs with fracture of the pelvis.

Symptoms are those of pain, hemorrhage and urinary extravasation.

Drainage of the extravasated area followed by repair of the urethral tear gives the best results. Use of an indwelling catheter increases the danger of infection. All cases require long after-treatment with sounds to dilate the subsequent stricture.

Impotence frequently occurs following this injury.

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SUBACUTE PERFORATIONS OF PEPTIC ULCERS*

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THE author's interest in this problem was aroused by a review of his diagnostic errors for the past two years and finding 4 cases in which he failed to recognize the existance of a subacute perforation of a peptic ulcer in the early stages. These patients all recovered and the diagnosis was subsequently made either by radiography or operation.

Most of us have been taught the clinical picture of the classical acute perforation of peptic ulcers; we also are familiar with the hyperacute or fulminating type of perforation. On the other hand, many of us have not realized that subacute or minor perforations do occur with considerable frequency and are accompanied by definite findings. Singer and Vaughan[1] were the first in this country to call attention to these subacute perforations and in 1930 gave them the name of "formes frustes." Since then reports of such cases have appeared by Eiskamp and Blaisdell,[2] Habein[3] and several others. In 1932, Singer and Vaughan[4] reported a further study of this problem.

Peptic ulcer is a fairly common condition; postmortem records show that ulcers of the stomach or duodenum are found in 4.5 per cent of individuals coming to autopsy. They are more common in males than females, the ratio being about 4:1. Statistics vary as to the occurrence in the various age groups but the ages thirty to fifty years furnish about 50 per cent of the ulcer cases.

Perforation is to be expected in about 10 per cent of the cases of peptic ulcer. Eliason and Ebeling[5] made a careful study of 729 cases of peptic ulcer. They found that approximately 75 per cent of the ulcers were located in the duodenum and 25 per cent in the stomach. In this series, 11 per cent of the duodenal ulcers and 7.6 per cent of the gastric ulcers perforated. This would give an average of about 10 per cent perforations for the entire group.

Commonly perforation of a peptic ulcer takes place into the general peritoneal cavity although it may occur into a walled off area or into the lesser omental bursa or into neighboring organs. Shelley,[6] in a study of 82 cases of perforation at St. Luke's Hospital in New York, found that perforation into the free peritoneal cavity occured in about 62 per cent of the cases; in 30 per cent the perforation was sealed or walled off and in about 8 per cent the perforation was retroperitoneal or into the pancreas.

Some perforations occur in the absence of any history suggesting previous ulcer symptoms. The great majority of the perforated cases, however, do give a long standing history of indigestion. Gilmour and Saint[7] in a study of 64 cases of acute perforation, state that in 88 per cent there was a history of indigestion for years. In the series presented by Shelley[6] there was a history of indigestion of more than two months duration in 91 per cent of the cases. Eliason and Ebeling,[5] in their extensive survey, found an absence of such a history in 18 per cent of the reported cases. Personal experience would seem to indicate however, that perseverence in searching for apparently minor details would be rewarded by a suggestive ulcer history in close to 100 per cent of cases. So frequently, the patient with a perforated ulcer is quite ill and the condition so obvious that we or our assistants do not feel justified in spending sufficient time to elicit these details.

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The acute perforation of a peptic ulcer is characterised by a fairly definite clinical picture. In the great majority of instances, the patient will give a history that his ulcer has been more active than usual in the past few days; he will tell you that there has been more pain and perhaps some vomiting. With the onset of acute perforation, there is commonly severe agonizing pain, located in the epigastric region. The pain is generally of the steady boring or burning type; the patient holds himself in a fixed position, lest any movement increase the severity of the pain. Occasionally we do see the exceptions to this however and the patient turns and twists with a colicky type of pain. About one-half of the patients with an acute perforation vomit with the onset and continue to retch and gag long after the stomach is emptied. The shock of the perforation is often so severe that the patient becomes cold and clammy and beads of perspiration stand out on the forehead. As one approaches such a patient, his pallor is noted, there may be some cyanosis of the lips and he really looks sick. In the early stages, there is tenderness in the subcostal angle.

Livingston in his monumental work "A Clinical Study of the Abdominal Cavity and Peritoneum," has clearly pointed out the reason why pain from a perforated ulcer is always referred to a definite location. Usually, there is spasm of the abdominal muscles amounting to almost board-like rigidity. This will be more marked about the level of the umbilicus in the early stages, before widespread peritoneal involvement occurs. There are other signs of acute perforation but these only are cited for comparison with the signs found in the subacute perforation. As involvement of the peritoneal cavity increases, the symptoms and physical signs rapidly become those of a general peritonitis and usually death ensues unless surgical intervention is successfully accomplished.

In the hyperacute or fulminating type of case, the symptoms are similar to those of the acute perforation but more severe and widespread; general peritoneal involvement is rapid and collapse occurs promptly. The duration of the process is measured in hours rather than by days as in the acute type.

Subacute or minor perforation is a much milder affair than the acute. Usually the actual perforation is preceded by a period of increased ulcer activity; often the perforation itself is considered to be merely a manifestation of the increase of activity. There may be pain and tenderness in the epigastrium; there is absence of the severe pain and shock seen with acute perforation. There may be vomiting but this is usually of short duration and often by the time the physician sees the patient, he finds the latter taking fluids by mouth with no apparent discomfort. Within a few hours after the onset, the patient appears quite comfortable and exactly what has occurred may be a matter of speculation. A great percentage of these minor perforations go on to spontaneous recovery and the patient is apparently completely well in three to four days. That there has been a subacute perforation may be proved by the finding of free gas in the peritoneal cavity by fluoroscopic examination in the early stages; later the diagnosis may be established by operation or radiography. The mildness of the symptoms following minor perforations may be explained upon the basis of a small amount of gastric contents at the time, a limited amount of leakage and the ability of the peritoneal surfaces to cope with such foreign material. It is true that many of the minor perforations are promptly plugged by mucus or sealed by omentum; this does not occur in all instances however; occasionally subsequent radiography or operation demonstrates that the perforation is still patent. The size and position of the ulcer apparently have little to do with the severity of the symptoms resulting from perforation. Waltman Walters, in discussing Habein's[3] case, says, "The size, position and extent of the inflammation is often out of all proportion to the duration and de-

gree of the symptoms." Shelly[6] in commenting on this question states, "The size of the perforation and the amount of induration apparently bore no relation to the history and physical findings."

Subacute perforations of peptic ulcers are very commonly mistaken for other conditions. Singer and Vaughan[1] state that about 2 of 3 of these cases are erroneously diagnosed when first admitted to hospital. The symptoms are quite frequently thought to be due to gastritis, pleurisy, central pneumonia, coronary heart disease, gall-bladder disease, etc.

From the literature on peptic ulcer one gathers the impression that subacute perforations are relatively infrequent. The fact is, that they are frequent enough but we fail to recognize them. In their study of this problem, Singer and Vaughan[1] found that 14 cases of subacute perforation in comparison to 12 cases of acute perforation, were admitted during a three months period at Cook County Hospital. This would seem to indicate that the frequency of these two types of perforation is about equal.

Treatment of the acute perforation of a peptic ulcer is based upon immediate operation at which time the ulcer is excised or destroyed by cautery, the ulcer bearing area inverted and the suture line covered with a tab of omentum; in some hands, a gastroenterostomy is also done at the same time. Most surgeons feel that acute perforations are surgical emergencies and that unless operated upon promptly, patients soon become moribund. Without operation at least 85 per cent of these patients will die.

What then is to be the method of treatment of the subacute perforation? To be logical, the same method is indicated. On the other hand, it is an established fact that a very considerable percentage of the patients with subacute perforations do recover without the benefit of surgery. Singer and Vaughan[4] reported a series of 40 such cases which occurred in a period of eighteen months at the Cook County Hospital. However, there are some instances in which surgery is postively indicated if the patient is to recover. Where the perforation closes spontaneously and there is mild peritoneal reaction, probably the patient will recover without surgery. The crux of the dilemma is to decide if the perforation is closed or not. This may be almost impossible. A safe rule is to operate upon all cases within the first twenty-four hours, where a diagnosis of perforation is established. If more than twenty-four hours has elapsed since perforation occurred and the process is apparently quiescent, operation should be deferred for it will only stir up infection and lead possibly to a fatal peritonitis. If, on the other hand, there is evidence of spreading peritoneal irritation, operation is indicated even though more than twenty-four hours has elapsed since perforation.

CASE REPORTS

Case 1. H. J., male aged forty-seven years, entered the hospital complaining of nausea and vomiting of forty-eight hours duration. One hour before admission he had been seized with a severe pain in the epigastrium which doubled him up and had continued since.

There was a history of bilious attacks, consisting of nausea and vomiting, occurring frequently for several years; no pain had been associated with these attacks.

Examination showed a thin individual who was apparently not in great pain. The temperature was 98.4, pulse rate 52, respirations 18. The chest was negative. The abdomen was scaphoid in shape; there was moderate rigidity throughout with tenderness in the epigastrium. At this time the white blood count was 12,240 with 38 per cent segmented forms, 43 per cent stab cells, 4 per cent juvenile forms and 15 per cent small lymphocytes. A waiting policy was adopted.

Eight hours later, the temperature was 100.2, pulse rate 80 and respirations 20. The patient was complaining of pain in the right shoulder blade but appeared comfortable; he volunteered the information that he felt much better. There had been no further vomiting. There appeared to be some retraction of the abdominal muscles although they moved quite freely with respira-

tion. Some spasm and rigidity of both sides of the abdomen was noted above the level of the umbilicus and there was slight tenderness in the right upper quadrant. Auscultation of the abdomen revealed no peristaltic sounds. At the end of another eight hour period, the temperature had increased to 101. F., the pulse rate was 82 and the respirations 20. The patient stated he felt much better although examination of the abdomen showed little change.

At the end of the twenty-four hour period, the temperature and pulse were as before but there was a very marked subsidence of the abdominal signs. At the end of forty-eight hours the patient showed further improvement and was taking fluids freely by mouth without apparent distress. At the end of seventy-two hours, the patient stated that he was "all right." His temperature was 98.6 F., pulse rate 70 and respirations 18. Nothing abnormal was noted upon examination, except slight tenderness in the epigastrium Treatment from that time on consisted of the usual ulcer regime. Fourteen days after admission, a gastrointestinal study showed definite ulceration of the duodenum with considerable pyloric spasm and retention of the meal.

Case II. J. M., male, aged forty-four years, entered the hospital complaining of severe upper abdominal pain of four hours duration, accompanied by retching. He stated that he had suffered with indigestion for more than one year, often had severe attacks and occasionally vomited blood in an attack. To him the present episode seemed just like several previous attacks. The temperature was 99.6 F., pulse rate 110, respirations 20, blood pressure 118/78. There was marked spasm of the upper abdominal muscles and generalized abdominal tenderness. The white blood count was 10,020; the differential count showed 59 per cent neutrophiles, 1 per cent myelocytes, 37 per cent small lymphocytes, 1 per cent endothelial cells and 2 per cent eosinophiles. Under liberal doses of morphine and hot packs to the abdomen, the acute episode rapidly subsided and within twenty-four hours following admission the patient was taking fluids freely by mouth with no apparent discomfort and presented no physical signs except slight epigastric tenderness. Three days later, a diagnosis of localized abscess was made but in view of the clinical improvement, drainage was not done at that time. Two weeks after discovery of the abscess operation was done, the abscess drained and an open perforation of the anterior wall of the stomach was closed with suture. Operative recovery was uneventful but subsequent investigation of the patient revealed that he was still having "stomach trouble."

Case III. J. K., male, aged sixty-three years, was seen with his physician because of severe abdominal pain of two hours duration; there had been no vomiting. The past history disclosed no complaint of indigestion but there had been mild attacks of dyspnea. The temperature was 97.8 F., pulse rate 60 and respirations 30. The heart was slightly enlarged to the right and the apical impulse diffuse; no rub was heard; there was an occasional premature contraction. The abdomen moved freely with respirations; it was not distended or retracted; there was some tenderness in the epigastrium accompanied by muscle spasm. The white blood count was 8220 with 73 per cent segmented forms, 18 per cent stab cells, 1 per cent juvenile cells, 1 per cent endothelial cells and 6 per cent small lymphocytes. The condition was considered to be a coronary one and medical treatment advised. On the day following, the patient was much improved; the temperature reached 100. F., with a pulse rate of 90 and respirations of 20; he was taking fluids by mouth freely. The abdomen was apparently negative. One week later, a gastrointestinal study showed definite evidence of an active duodenal ulcer. Subsequent cardiac examination including electrocardiography was essentially negative.

Case IV. F. C., male, aged forty-five years, entered the hospital complaining of severe abdominal pain and vomiting of two hours duration. He had suffered with indigestion for years and there had been frequent attacks of vomiting. The temperature upon admission was 98.4 F., pulse rate 110 and respirations 20. The pain was apparently of the colicky type and between seizures the patient moved about freely in bed. The entire abdomen was spastic with marked tenderness in the upper left quadrant. The white blood count was 13,900 of which 54 per cent were segmented forms, 2 per cent stab cells, 3 per cent juvenile forms, 1 per cent myelocytes, 1 per cent eosinophiles and 5 per cent small lymphocytes. The patient was given morphine freely, nothing by mouth and fluid was supplied by the intravenous route.

At the end of twenty-four hours, the patient stated that he had no pain; however there was some rigidity in the upper left quadrant of the abdomen, and the temperature climbed to 101 F. After three days in the hospital all evidence of the acute process subsided, the examination was negative and the temperature normal. One week later a gastrointestinal study was done which showed an irregular pylorus and a fair sized extragastric area which contained gas and filled with barium. Surgery was advised but refused and later the patient was discharged with no subjective signs.

CONCLUSIONS

1. Subacute perforations of peptic ulcers are not rare.

2. The subject of subacute perforations is discussed and the literature reviewed.

3. Four personal cases of subacute perforation are cited.

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INFRAMURAL GASTROSTOMY

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A PROCEDURE for gastrostomy should be technically simple and provide a permanent means of feeding the patient directly through the stomach, because many of these patients constitute the poorest risks.

Permanent good physiological function is essential because a gastrostomy that functions well for a short time only may become such a curse as to render the patient utterly despondent and disgusted.

Perhaps the most unsatisfactory feature of gastrostomy is leakage of the acid stomach contents.

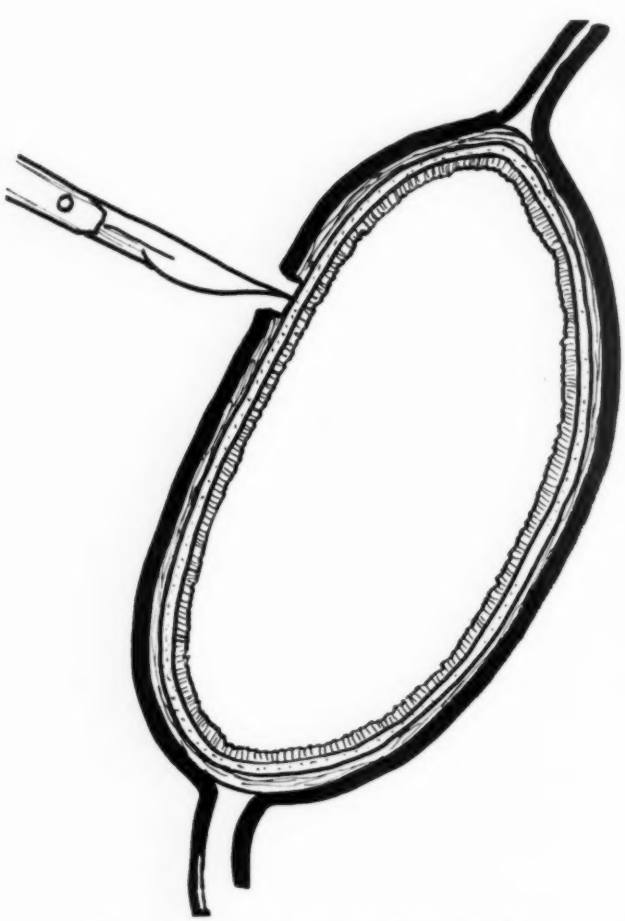

FIG. 1. Incision of serosa and muscularis.

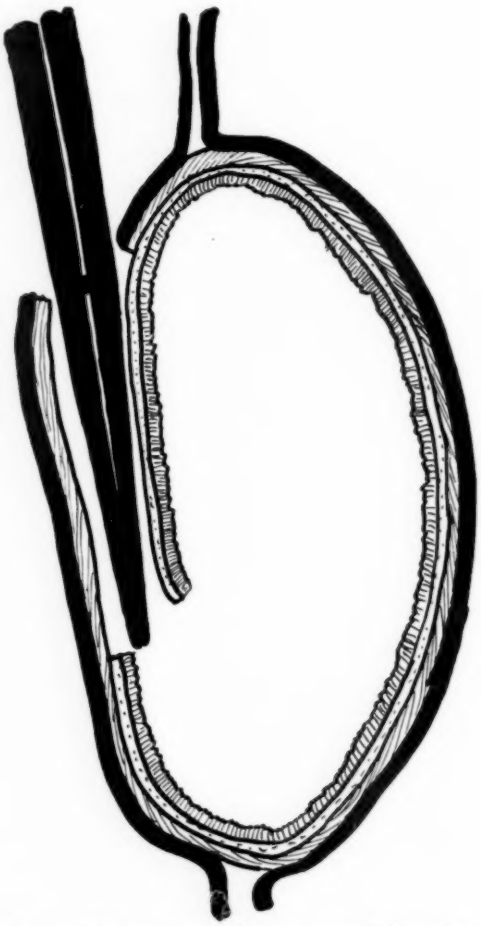

FIG. 2. Scissors creating a tunnel between muscularis and mucosa and making small hole in mucosa.

It might seem useless to present a new procedure for gastrostomy when there are so many excellent ones in use, but I present the inframural technique, hoping that it might be of real service.

The fundamental idea of the inframural gastrostomy is the burrowing of a long

narrow, perpendicular passageway for the catheter between the muscularis and the mucosa of the stomach. This long channel, the outer opening of which is close to the upper (small curvature) and the inner to the lower border (greater curvature) of the stomach prevents any spilling of stomach contents.

The procedure is extremely simple. Under local anesthesia the linea alba is incised for about two inches midway between the umbilicus and the xyphoid. The stomach is exposed and raised. As near the border of the small curvature as practical, the serosa and muscularis are incised exposing about one-eight of an inch of mucosa (Fig. 1). A blunt pair of scissors is then forced between the exposed mucosa and the muscularis and a narrow tunnel between these two stomach layers is created by pushing the closed scissors downward toward the greater curvature until their points reach the spot in which the surgeon intends to make the inner opening (Fig. 2). A small hole is then made in the mucosa by pressing the points of the scissors on it and opening and closing them slightly, while they press on the mucosa itself. The scissors are withdrawn and a catheter of appropriate size is introduced in the tunnel thus created, until it is felt emerging within the stomach.

Fig. 3. Relationship of the laparatomy incision, L, and the stab wound incision, C, for the passage of the catheter.

A stab incision the size of the catheter is made through the abdominal wall at about 2 cm. to the left of the laparatomy incision (Fig. 3), through which the catheter is carried out of the peritoneal cavity.

Two sutures of No. 2 chromic catgut are then placed on the stomach at the site of the upper incision (Fig. 4). These two sutures are intended to secure the catheter in the stomach and cause adhesions between the parietal peritoneum and the stomach serosa at the points where the catheter emerges through the wall of the stomach and is carried out through the abdominal wall (Fig. 4). The upper suture is placed about 0.5 cm. above the stomach incision and the lower suture about 0.5 cm. below the same incision.

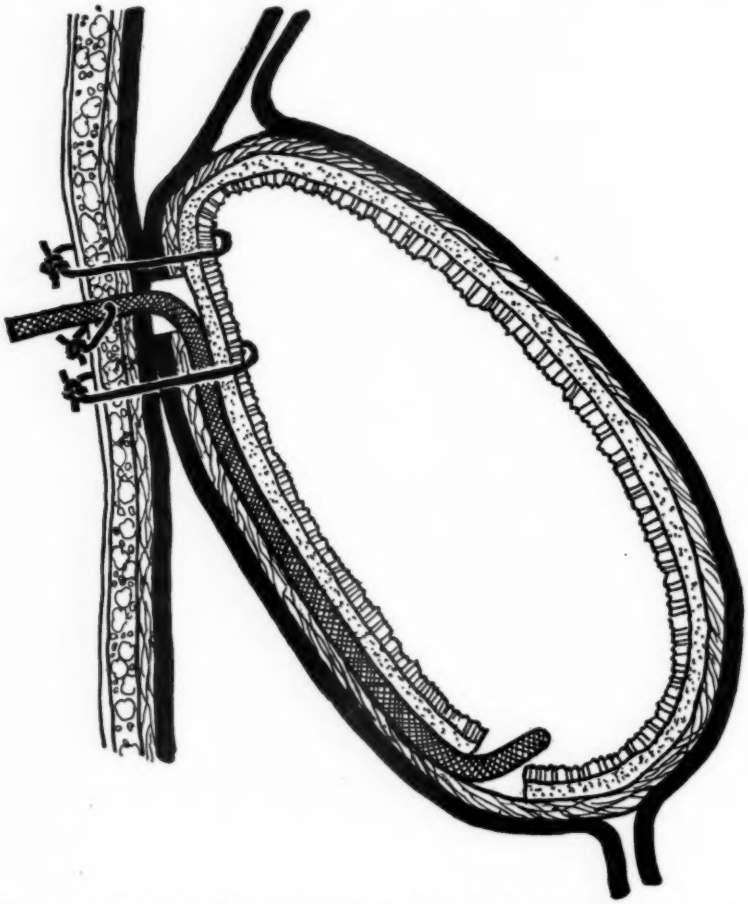

Fig. 4. Catheter passed into stomach through the tunnel between the muscularis and the mucosa. Stitches securing a small portion of the stomach to the parietal peritoneum and catheter are shown in position.

Both sutures go through the whole thickness of the stomach, the needle entering and emerging a few millimeters beyond the incised stomach serosa. Both ends of each suture are separately brought out through the abdominal wall and tied. The peritoneal coat of the stomach around the stomach incision is thus made adherent to the parietal peritoneum while the passage-way for the catheter is excluded from the abdominal cavity with very little area of the stomach itself disturbed. Although the catheter is secured in position by the lower suture (Figs. 3 and 4) it is advisable to add a stitch between the catheter and the skin. The middle incision is then closed in the usual manner.

The patient may be fed immediately.

A small separate stab wound for the passage of the catheter is advised because the laparotomy incision is thereby allowed to heal undisturbed by primary union.

The advantages of the inframural gastrostomy are:

Simplicity and rapidity of performance under local anesthesia; no shock; possibility of feeding the patient immediately; minimal deformity of the stomach; minimum of adhesions; formation of a leakage proof stoma.

CONCLUSIONS

A method of producing a long narrow passageway for the feeding catheter through the stomach wall is described. This passageway besides being long has two openings which are situated perpendicularly at the two extremes of the stomach itself. This arrangement prevents leakage of the stomach contents because fluids cannot travel against gravity and the physiological function of the stomach is left practically undisturbed.

It may be emphasized that neurosurgical experience has thoroughly justified the anatomical concept of the pathways taken by the stimuli which result in cardiac pain . . . Attacks of angina pectoris have invariably stopped after thoracic ganglionectomy or section of the upper thoracic posterior spinal roots, but experience has demonstrated that these operations are dangerous and usually unnecessary. Alcohol injection is safe and, in the hands of an expert, highly successful. It cannot be expected, however, that the occasional operator can inject the upper thoracic ganglia and their rami with uniform success.

Diseases of the Coronary Arteries and Cardiac Pain Edited by

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USE OF PITRESSIN FOR CONTROL AND RELIEF OF DISTENTION*

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THE use of pituitary preparations for the prevention, control, or relief of postoperative abdominal distention was first mentioned nearly thirty years ago. Since that time a good many reports on the use of pituitary extracts have appeared in the literature, a large number of them lauding the satisfactory results which have been obtained.

It is the twofold purpose of this paper first, to analyze the available experimental and clinical data, and second, to report the results in a series of patients treated with a potent pituitary preparation.

In 1909 Dale[1] first reported that the action of posterior pituitary lobe extracts was one of direct stimulation of the involuntary musculature throughout the body without relation to innervation. In the same year Blair Bell[2] observed the action of an intramuscularly injected pituitary extract on the intestinal tract of pithed rabbits and noted violent peristalsis followed by the expulsion of feces. Accordingly he used it in several cases of postoperative abdominal distention. His report is the first describing its use clinically for the relief of this condition and he stated that the results were generally good in that the extract induced the expulsion of flatus and feces.

Since that time there has been a great deal of experimental and clinical investigation in an effort to determine, (1) the true action of various pituitary extracts on the intestinal musculature, and (2) their value clinically. Unfortunately for the clinician, the laboratory has supplied him with a mass of contradictory evidence. This is shown by a review of the literature on this subject.

The experimental data can be roughly divided into two main groups, the one attempting to show that the injection of pituitary extract produces either an increase in tone or increased peristalsis, the other indicating that relaxation is the usual effect produced.

To the first group belong the experiments of Franchini[3] who noted severe intestinal contractions with diarrhea in dogs following the injection of extracts of the whole pituitary gland, prepared in his own laboratory. He occasionally found an intense enteritis with submucosal hemorrhages after the injections, usually after large doses had been used, or when the extract had been administered intravenously. Young,[4] using isolated strips of ileum from cats substantiated the contractile properties of the extract, although he remarked on the variability in the degree of this action. Dixon[5] reported a uniform increase in the tone of isolated loops of cat intestine, including portions of both small and large bowel, after the injection of whole gland extract. Using strips from the muscularis of the stomach of cats, dogs and rabbits, McSwiney and Brown[6] likewise observed a constant increase in tone when posterior lobe extract was added to the bath in which the strips were immersed. Boenheim's[7] experiments with posterior lobe extract resulted in strong peristaltic contractions in both the small and large bowel. Gorke and Deloch[8] were the first to study these reactions roentgenologically. After giving their sub-

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jects a barium meal, 1 c.c. of posterior lobe extract was injected subcutaneously and the gastrointestinal tract observed with the fluoroscope. The stomach showed violent contractions, but emptied slowly due to pylorospasm. In the remainder of the intestinal tract they observed an increase in tone and stronger peristalsis.

In the middle ground of the controversy of stimulation versus relaxation stands the work of MacDonald[9] whose experimental results led him to state that although the general effect of posterior pituitary extract was one of stimulation if large enough doses were given, the degree of activity produced was by no means uniform. Shamoff[10] was led to somewhat the same conclusions by his experiments in which he employed several standard commercial preparations of all three parts of the pituitary gland. He was the first to report that the extracts of the anterior lobe and pars intermedia were totally inactive on the smooth muscle of the intestine while that of the posterior lobe produced a wide range of effects on isolated intestinal strips, varying from marked stimulation to definite relaxation. These same preparations, however, showed a uniformity of diuretic and hemodynamic action.

The chief protagonists of the relaxation concept are Voegtlin and Dyer[11] who noted nothing but relaxation in strips of rat jejunum immersed in solutions of posterior lobe extract of varying concentrations. Similar results were reported by Hoskins[12] who suggested that the reason for his failure to obtain the stimulation found by others may have been some substance used in the preparation of his extracts. It remained for Roth[13] to prove this point by showing that extracts prepared with chlorbutanal or chloretone produced intestinal relaxation while his own preparations, free from these substances, caused an increase in tone. Evidence has also been brought forward to show that the diet[14] and the emotional state[15] of animals from whom the gland is removed for extraction has a definite influence on the type of reaction which the extract produces.

Geiling[16] in his review published in 1926 summed up the then existing thought on the action of posterior lobe extract by stating that though some recent work showed a tendency toward relaxation the main evidence was in favor of stimulation of the gastrointestinal musculature. He considered the method of preparation as an important factor in determining the type of effect which the extract will produce, and besides the factors already mentioned suggested that excessive acidity might result in a "relaxing" instead of a "stimulating" extract. Kauffman,[17] however, shortly thereafter, demonstrated that alkalinization produced no change in the stimulating action of his preparations, but was able to show, that by varying the *p*H during his extractions, the fraction which caused intestinal contraction was different from that which raised the blood pressure and stimulated the uterus. It was not long after this that Kamm[18] and his co-workers isolated two active principles from the posterior lobe of the pituitary gland, a pressor and an oxytocic fraction. They made no attempt to determine the various actions of these principles on the intestinal musculature. Elmer, Ptaszek and Scheps[19] demonstrated that the two fractions had separate actions on the intestinal musculature which were antagonistic, the pressor fraction stimulating, the oxytocic relaxing. Their work further showed that when injected together, the effect of one was neutralized by the effect of the other. This was further illustrated by the fact that the vasopressor fraction produced much greater stimulation than pituitrin, which, of course, contains some of both principles.[20] These findings have recently been confirmed by Melville.[21] This, therefore, serves in part to explain the variability in the activity of the older extracts of the posterior lobe, which contained varying amounts of both fractions—pressor and oxytocic.

Most of the related experimental work done since that time tends to confirm the earlier concept, namely, that the pressor fraction of posterior lobe extract has a very definite stimulating action on the normal intestinal musculature. Ample evidence of this is found in the work of Carlson,[22] Robson,[23] Gruber and Robinson,[24] Krishnan[25] and Elmer, Ptaszek, and Scheps.[26]

The only results which do not fall in line with this concept were some obtained in abnormal states of the intestinal tract. MacIntosh and Owings[27] showed that the vasopressor fraction caused a fall in tonus and a decrease in peristalic activity in the small intestine of dogs where complete mechanical obstruction had been obtained by transection of the lower ileum. They made their determinations by measuring pressure changes in a balloon placed within the bowel just above the site of obstruction. Using the same balloon technique in isolated closed loops of both small and large bowel, Quigley and Barnes[28] also noted a constant decrease in tone and motility after use of the pressor fraction. These results are understandable and the distinction between the effect of the pressor fraction on normal and abnormal bowel is a valid one. Melville and Stehle[29] isolated special pressor and oxytocic fractions for their own use and compared their action on isolated loops and on the normal gastrointestinal tract observed by fluoroscopic examination after the ingestion of barium. With the pressor substance they observed uniformly an increase in intestinal motility with contraction often followed by defecation. The isolated loops, however, showed no clear cut effect. In all instances the action of the pressor substance was identical with that of commercially prepared pitressin; their oxytocic fraction and commercial pitocin gave constant evidence of relaxation.

The preponderance of evidence, therefore, points to the fact that the pressor fraction of the posterior lobe of the pituitary gland exerts a stimulating effect on gastric and intestinal musculature under normal conditions. That this effect is variable in the intensity of its action is not more difficult to understand than the variability of other hormone actions. No definite statement can be made as to the degree of activity of the pressor fraction on the different parts of the gastrointestinal tract.

While the investigative work was being carried on in the laboratory, clinicians were experimenting with the action of pituitary preparations on abdominal distention in the patient. The first report of its use, following Blair Bell's[2] original article, was in a symposium on the treatment of generalized peritonitis. Renton[30] reported that he had obtained satisfactory results with intramuscular injections of pituitary extract both before and after drainage operations. The extract not only increased peristalsis but also brought his patients out of shock. The several other articles and the subsequent discussions failed to disclose any further information on the subject, showing that the use of this substance was still in the experimental stage. The following year (1911) Bidwell[31] discussed his results after the routine postoperative use of pituitary extract in 21 unselected cases on whom laparotomy was performed, all involving excessive trauma, or infection. One cubic centimeter of the extract was injected every six hours for twenty-four to thirty-six hours. In all but one patient there resulted the early passage of flatus, absence of distention or of marked abdominal discomfort. These patients all had remarkably slow pulse rates and no untoward reactions of any kind were observed. It is interesting to note that one woman four and a half months pregnant was carried through such a regime without mishap.

One other article[32] appeared at this time advocating the use of pituitrin in peritonitis but emphasis was placed on its hemodynamic action.

During the next three years a series of papers were published from different coun-

tries reporting favorably the use of various pituitary preparations in postoperative abdominal distention with, and without, infection of the peritoneum.[33–38] In all, 59 cases were reported and emphasis was placed on the consistent ability of pituitary preparations to produce peristalsis and the expulsion of flatus and feces, where all other measures failed. In the majority of patients the preparation was given hypodermically and no unpleasant effects were recorded. One review,[34] however, mentions the dangers of administering it intravenously and describes the intense pallor, rapid irregular pulse, shallow respiration, semistupor and intense frontal headache which occasionally follow this method of administration.

There follows a period of about ten years during which mention was rarely made of the use of any pituitary extract in abdominal surgery.

In 1924, Blake[39] reported favorably on the use of pituitrin in peritonitis and his article was followed shortly by Mayer's[40] description of the intravenous use of a similar preparation in 52 cases of peritonitis complicated by severe ileus. Four to six cubic centimeters were given in 500 to 1000 c.c. of physiological saline solution. Twenty per cent of all the cases survived, this number comprising about 30 per cent of the cases in which the ileus was relieved by the drug. All patients who were not relieved by the extract succumbed. A comparable percentage of recoveries from peritonitis (22 per cent) in a series of 81 cases treated in a similar manner was reported by Vogt.[41]

Potter and Mueller[42] then published results on the prophylactic treatment of postoperative abdominal distention with pituitary preparations. One hundred patients received 1 c.c. of surgical pituitrin every four hours for six doses. Distention rarely developed and then only in those patients who were distended preoperatively or after the pituitrin had been discontinued. In 1928, after Kamm et al. had isolated the pressor fraction another series of 100 patients were treated with 8 to 12 postoperative injections of 1 c.c. of the pressor fraction at four hour intervals. Of this group only 2 developed any distention and no untoward effects were observed.

In view of their results and three other subsequent favorable reports on the use of pitressin[26,43,53] from German and British clinics it is strange that a review of the American literature on the treatment of distention with or without peritonitis during the past five years reveals an almost unanimous feeling of condemnation of the use of pitressin or similar substances.[44–51] The chief reasons given against its use are (1) that its temporary stimulating effect is followed shortly by more complete relaxation, (2) that it only affects the large bowel, which is rarely involved in postoperative ileus, and (3) that it produces stimulation of the bowel, when rest is indicated. Another author on the subject feels that "the momentary (beneficial) effect is a negligible violation of the principle of rest" in peritonitis.[43] There is, of course, ample experimental evidence to show that the small bowel is as actively affected by the pressor fraction as is the colon.

More recently Rundle[54] has reported on the intravenous use of pitressin. He used 0.5 to 1 c.c. injected very slowly; 6 cases were so treated, 4 clean and 2 with peritonitis. None showed any untoward reaction and the results in all were prompt and dramatic with the passage of feces and flatus, in some cases even before the injection was completed.

In summing up the status of the clinical use of pituitary preparations several rather interesting points are prominent. In the first place after a period of nearly thirty years during which they have been used in many clinics the world over, there is no unanimity of opinion as to their value. Furthermore, no explanation has been offered for the marked variations in their effectiveness. This, of course, includes the period prior and subsequent to the isolation of the vasopressor fraction of the

posterior lobe. Before its isolation extracts from the whole lobe were used and there is adequate explanation for the lack of uniformity of effect of these preparations, in the proved antagonistic action of the two fractions. Since that time, however, only the pressor fraction has been employed.

The second outstanding fact is that, while British and German authors continue to report satisfactory results, American surgeons have not only ceased to use the substance except in occasional cases, but also have unanimously condemned its use in patients where the distention is associated with peritoneal infection. The reason for this state of affairs is not obvious unless possibly it implies that there is some essential difference between the commercial preparations here and abroad.

A final interesting fact is that, although almost everyone who has written on this subject has mentioned the possible dangerous reactions which may occur after the injection of pitressin, no one has reported a single case which was seriously or critically affected by such a reaction, even after intravenous administration,[54] and not one death has ever occurred. Since the incidence of these reactions is so low and their effect, as a rule, so mild and transient, it does not seem probable that they can have played any part in influencing any one against the use of pitressin.

In an effort to determine at first hand the value of pitressin in postoperative abdominal distention and its associated symptoms, the substance was administered to a series of 94 patients on the surgical service of Dr. I. S. Ravdin at the Hospital of the University of Pennsylvania. The results were carefully observed and recorded on a specially prepared sheet on the patient's chart. The amount given subcutaneously varied from 0.5 to 1 c.c. every four hours given for as long as ninety-six hours. The series includes a group to whom it was given routinely after operation without waiting for symptoms to develop, another group who received pitressin to relieve distention and gas pains, a third small group to whom it was given to control severe and obstinate distention due to ileus from some other cause than operative trauma or infection and finally a group with widespread peritoneal infection.

The various types of operation which these patients underwent are listed below. The results can best be shown in Tables I, II, III, IV, V and VI.

TABLE I

No operation	4
Cholecystectomy	8
Cholecystostomy	3
Appendectomy	30
Appendectomy—drained	12
Herniorrhaphy	8
Lysis of adhesions for obstruction	7
Pelvic operations	3
Ileostomy and cecostomy	7
Closure of perforated ulcer	3
Intestinal resections	2
Splenectomy	4
Gastroenteroenterostomy	3
Total	94

TABLE II

Total No. of Cases, 24

Patients given Pitressin by Routine Postoperative Administration	No.	No Symptoms	Patients with Distention	Patients with Gas Pains
0.5 to 1 c.c. every four hours	24	17	4	3

TABLE III

	Patients given Pitressin Postoperatively for Distention				Patients Who Passed		Marked Increase in
Degree of Distention	No.	Improved	Unimproved	Made Worse	Gas	Feces	Peristalsis
Minor	9	6	3	0	5	1	4
Moderate	38	37	1	0	26	10	10
Marked	19	14	5	0	13	6	8

In the entire series only 3 patients suffered reactions following the injection of pitressin. These were characterized by in-

tense pallor, headache, shallow respirations and a rapid thready pulse. All were transient, lasting from ten minutes to one hour, and the patients recovered spontaneously without the administration of any stimulants.

Table IV

Patients given Pitressin Postoperatively for Gas Pains

Severity of Gas Pains	No.	Improved	Unimproved	Made Worse
Minor	7	7	0	0
Moderate	25	21	4	0
Marked	15	14	1	0

Table V

Cases with Distention Unoperated	No.	Improved	Unimproved
	4	3	1

Table VI

Cases with Widespread Peritonitis	No.	Distention Improved	Distention Unimproved
Recovered	10	10	0
Died	5	3	2

One other fact must be recorded. Blood pressure readings were determined on the majority of these patients just before and shortly after one or more injections. We were able to find no marked rise or fall in either systolic or diastolic pressure following the injection of pitressin in any case.

In our experience, pitressin has proved a valuable agent in the prevention and relief of abdominal distention due to adynamic ileus resulting from operative trauma, and peritoneal infection. Reactions are few, mild and transient and associated with no danger to the patient. Blood pressure is apparently not affected. We, therefore, do not hesitate to recommend its use in combatting this distressing postoperative symptom.

SUMMARY

1. The experimental and clinical literature on the effect of posterior pituitary lobe preparations on the gastrointestinal tract is reviewed and analyzed.

2. A series of 94 cases treated with pitressin is reported and the results tabulated. Relief from symptoms was obtained in the majority of cases.

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MALUNITED FRACTURES OF LOWER END OF HUMERUS*

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THE patient who has a malunited fracture of the lower end of the humerus seeks the advice of the orthopedic surgeon because of a stiff and unsightly elbow. The deformity and disability usually are due to union of the fragments in faulty alinement. The poor result may have followed a circulatory disorder in the forearm and hand which accompanied the fracture and necessitated a delayed reduction, or it may have been due to inexperience on the part of the attending doctor; however, malunion and functional disability may occur even in the hands of the most skillful fracture surgeon. This paper is presented not as an original contribution but as a review of the commoner methods of management of malunited fractures of the lower end of the humerus and as a short comment upon their most frequent complications.

The elbow is an intricately fashioned joint. After fracture involving the articular surfaces, it is often very difficult to effect accurate reapproximation of the fragments, particularly in the case of comminuted intercondylar fractures. In the restoration of a joint following an old fracture, the primary object of treatment is to obtain improvement of its function; the secondary object is to improve its appearance, but function must never be sacrificed in any way for appearance. The hinge-like motion between the ulna and the lower end of the humerus is of greater usefulness than the rotary motion of the head of the radius upon the capitellum; however, a perfect result in the treatment of fracture about the elbow joint must include restoration of both of these motions.

The circulation about the lower end of the humerus is abundant. When non-union occurs it is never a result of inadequate blood supply but is due to unsatisfactory approximation of the fragments. Due to the frequency of stripping and tearing of the periosteum in these elbow fractures, excessive callus formation and calcification occur within the surrounding tissues and may lead to partial ankylosis. The amount of osteogenesis following bone injury about the elbow is in direct proportion to the degree of periosteal injury. Fractures with marked displacement are accompanied by the largest amount of new bone formation. The excessive callus may form a serious handicap to a good functional result.

In young children epiphyseal injury is an important lesion. Accurate approximation of the epiphyseal surfaces is often difficult to obtain, even by open operation. With subsequent growth a distortion of the joint often occurs, resulting in serious deformity and disability. In very young children the extent of the epiphyseal injury cannot be visualized accurately in roentgenograms because of the large proportion of cartilage present. In the interpretation of the roentgenogram care must be taken to differentiate between the epiphyseal line and a fracture line. A comparison with the roentgenogram of the normal elbow is helpful. Fortunately, epiphyseal separations are not so common as fractures.

In the reposition of malunited fractures the angle of the lower end of the humerus to the shaft must be considered. The normal elbow in extension has a carrying angle of from 6 to 16 degrees; i.e., the forearm possesses this amount of lateral deviation from the longitudinal axis of the upper arm. The carrying angle is dependent upon the contour of the lower end of the humerus and the upper end of the radius and the

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ulna. The articular surface of the lower end of the humerus forms anteriorly an angle of 45 degrees with the longitudinal axis of the shaft; this angle also must be considered in the reposition of these fractures. Accurate restoration of the carrying angle, while not essential to complete functional recovery, is necessary if a perfect anatomical result is to be obtained. In the restoration of the carrying angle, injury of the internal lateral ligaments is of importance, since relaxation of these structures tends to result in increased valgus.

Fig. 1. Old supracondylar fracture showing cubitus varus.

Any varus or valgus deformity of the elbow is accompanied by a change in the carrying angle and is spoken of as a gunstock deformity. The most common deformity is a cubitus varus, or loss of the normal carrying angle; it may occur following supracondylar fracture (Fig. 1). A cubitus valgus deformity most often results from fracture and displacement of the external condyle (Fig. 2). In addition to the gunstock deformity a bony projection may occur about one of the condyles and give the elbow an unsightly appearance. The healing of displaced intercondylar fractures is often accompanied by considerable broadening of the lower end of the humerus.

Five types of malunited fracture of the lower end of the humerus lead frequently to functional disability: (1) supracondylar, including diacondylar fracture and separation of the lower humeral epiphysis, (2) external condylar, (3) internal condylar, (4) intercondylar or comminuted "T" or "Y" fractures and (5) fractures involving the articular surface of the capitellum or trochlea.

The treatment and prognosis of each of these five types of fracture will be discussed individually, but there are certain general considerations which apply to the entire group. Most authors believe that in operating upon these malunited fractures conservatism is most important. Cotton reports the observation that in a great many cases surgery is followed by increased bone proliferation and further loss of function, especially when an attempt is made to remove bone filling the coronoid or olecranon fossa or deposited about the external condyle.

Campbell has enumerated the following five objects of reconstructive operations upon the lower end of the humerus: (1) reconstruction of a normal contour and alinement of the elbow joint surfaces, with

especial reference to the carrying angle; (2) improvement or complete restoration of motion; although mobility is desirable, however, stability or efficient function should not be sacrificed for the mere restoration of motion; (3) preservation of the condyles with their articular surfaces, when possible, and the promotion of union between the detached condyles and the shaft in as nearly the normal relationship as possible; (4) in "T" fractures, union and proper alinement between the condyles themselves and between the condyles and the shaft; and (5) excision of bony blocks or bridges that prevent full motion. When the disability is due to an excess formation of callus, or to a myositis ossificans, no operation should be attempted until ossification has become complete.

Ashhurst in 1910 stated that the secret of a good functional result is an early accurate reduction with fixation in hyperflexion. The position of hyperflexion favors preservation of the normal carrying angle, as the triceps tendon and muscle hold the fragments in position when the elbow is hyperflexed. Ashhurst contended that circulatory disturbances were unlikely to occur following complete reduction. It must be borne in mind, however, that hyperflexion cannot be carried to the point at which obliteration of the radial pulse takes place.

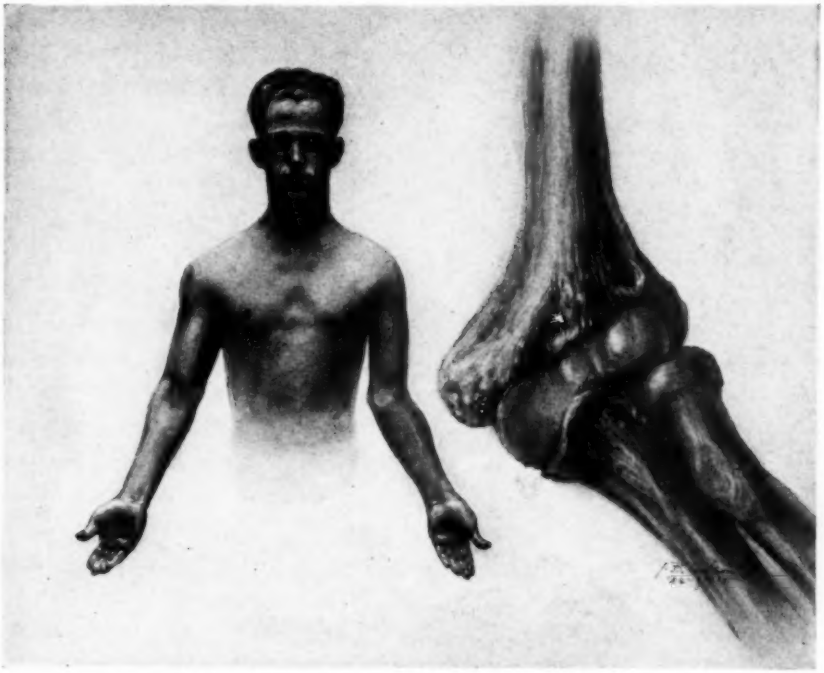

Fig. 2. Old supracondylar fracture showing cubitus valgus.

Eikenbary, among others, has advocated for these elbow fractures less physical therapy and more active motion. He feels that manipulation in an attempt to increase the range of elbow motion is often followed by tragic results.

SUPRACONDYLAR FRACTURES

In this group are included not only fractures above the condyles, in which the shaft takes a distinct forward bend, but also those through the condyles, or diacondylar fractures, and the traumatic displacements of the whole lower epiphysis. Separation of the entire lower epiphysis is very uncommon after four years of age. A very rare type of fracture which belongs in this group is that described in 1901 by Professor Posadas of South America; this is a diacondylar fracture with posterior displacement of the radius and ulna. This fracture practically always results in anky-

losis of the elbow, although Ashhurst reported one case in which a good functional result was obtained after a closed reduction.

Fig. 3. Supracondylar fracture in a boy, aged eight years (anteroposterior views); A, immediately after fracture; note medial and posterior displacement of lower fragment; B, three years later, showing cubitus varus deformity; function in elbow is normal; this fracture was never reduced. (*After Sever and Gallupe.*)

Supracondylar fractures are commonest in children whereas the external condylar fractures are more frequent in adults. Gilcreest reports that one-third of the fractures about the elbow joint are of the supracondylar type. He describes four varieties of supracondylar fracture: (1) the extension type is the most frequent; in this the fracture line passes from the anterior surface upward and backward, and the lower end of the upper fragment is displaced into the antecubital space; (2) the flexion type, which is the reverse of the extension type and in which the long upper fragment becomes displaced posteriorly; (3) the adduction type, and (4) the abduction type. Either of the two latter types may be associated with either of the two former. The flexion and extension supracondylar fractures were first described by Kocher.

In supracondylar fractures in children a good functional elbow with very little evidence of deformity may follow even marked displacement of the fragments (Figs. 3 and 4). This fact emphasizes the need for conservatism in the treatment of supracondylar fractures in childhood. Eliason says, "Do not pass on the result for at least a year. Children obtain good results, but patients past middle life get rather unsatisfactory function."

The most common types of malunion are: (1) a backward displacement of the lower fragment with the lower end of the shaft projecting into the antecubital space; (2) in addition to the backward displacement of the lower fragment there is a lateral or medial displacement and rotation, usually the inner side going posteriorly, resulting in cubitus varus; and (3) a forward displacement of the lower fragment, which also may be displaced medially and rotated.

Forward displacement of the lower fragment occurs in supracondylar fractures of the flexion type.

After solid union has taken place there may be limitation of flexion due to projection of the upper fragment into the antecubital space. It may be advisable to make a lateral incision and chisel off the projecting bone, as first described by Finney. Care must be taken to remove more bone than is absolutely necessary to permit full flexion.

Sinclair recommends that the spur which projects into the elbow joint should be removed by means of a burr attached to an electric saw. This is a very simple and effective method. This operation is not always indicated in children, as is exemplified by the case shown in Figure 5. As epiphyseal growth continues it is evident that the upper fragment grows away from the joint and at the same time the projecting bone is absorbed and becomes progres-

sively less prominent. After operative removal of bony spurs motion should be started early.

alinement (Fig. 6). If there is a bone fragment projecting into the antecubital space, this can be removed at the same time.

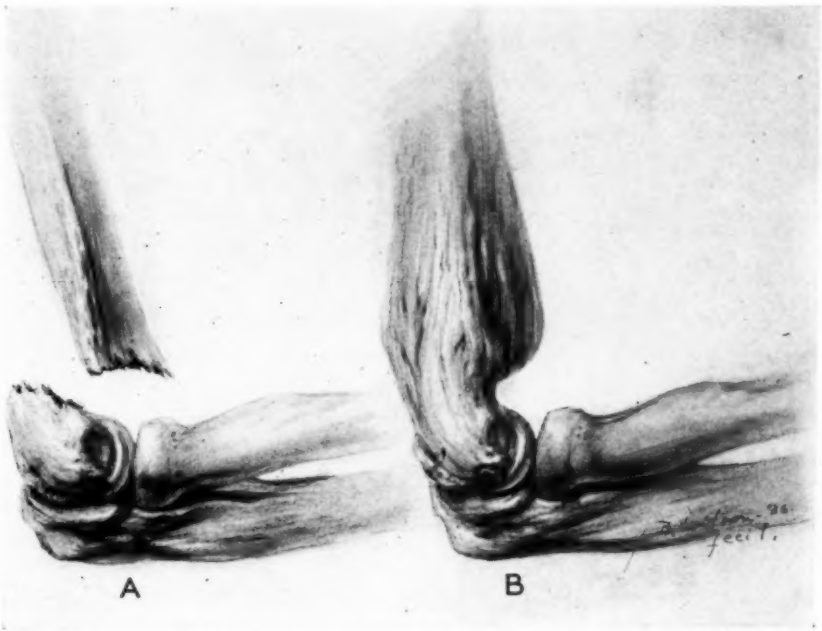

Fig. 4. Supracondylar fracture in a boy aged eight years (lateral views); A, immediately after fracture showing displacement of fragments; B, three years later; this fracture was never reduced. Function normal. (*After Sever and Gallupe.*)

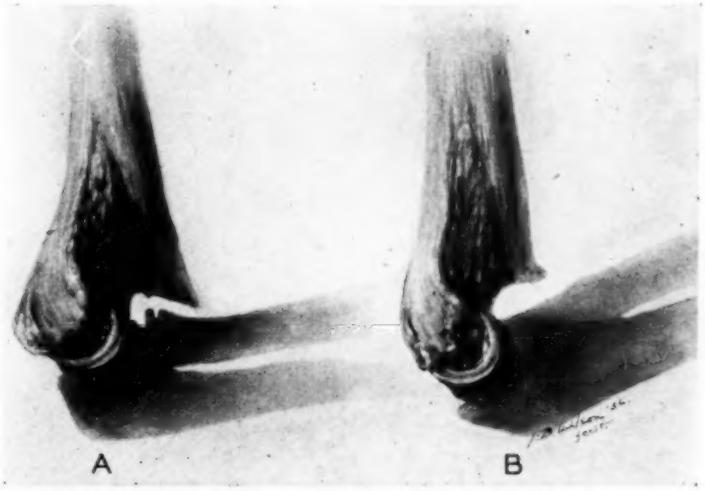

Fig. 5. Old supracondylar fracture showing absorption of projection into antecubital space; A, five months after injury; B, three years after injury. (*Courtesy of Dr. J. W. White.*)

If in addition the lower fragment is displaced to the medial side and rotated, producing a varus deformity, an osteotomy through the original fracture site may be necessary for the restoration of normal

The treatment is then that of a fresh supracondylar fracture of the extension type, the elbow being immobilized in hyperflexion. At times there is danger that the fragments may slip apart, and it

may be necessary to hold them together by inserting a bone peg through the lower into the upper fragment.

Fig. 6. Old supracondylar fracture with cubitus varus deformity; A, note position of wedge osteotomy; B, after osteotomy showing correction of varus deformity.

It may be necessary to remove bone which projects posteriorly following the malunion of a flexion supracondylar fracture. An osteotomy also may be indicated if there is rotation and medial displacement of the lower fragment. Occasionally the osteotomy is best done through a posterior approach, in which event the triceps muscle should be split.

Occasionally a marked anterior angulation follows supracondylar fracture (Fig. 7). In such cases anterior cuneiform osteotomy should be done. This will increase both flexion and extension of the joint. The postoperative treatment is that of a fresh supracondylar fracture.

The treatment of old unreduced epiphyseal displacements resulting in deformity is the same as that of supracondylar fractures.

Cohn has called attention to the development of the external rotators of the humerus in supracondylar fractures with cubitus varus deformity. He believes that if the external rotators of the humerus are not strengthened after the osteotomy and correction of the deformity, a tendency toward recurrence of the varus, of muscular rather than bony origin, will result. Cohn also believes that at times it is wise to put a drain into these joints after the osteotomy in order to prevent the excessive swelling which sometimes ensues. If swell-

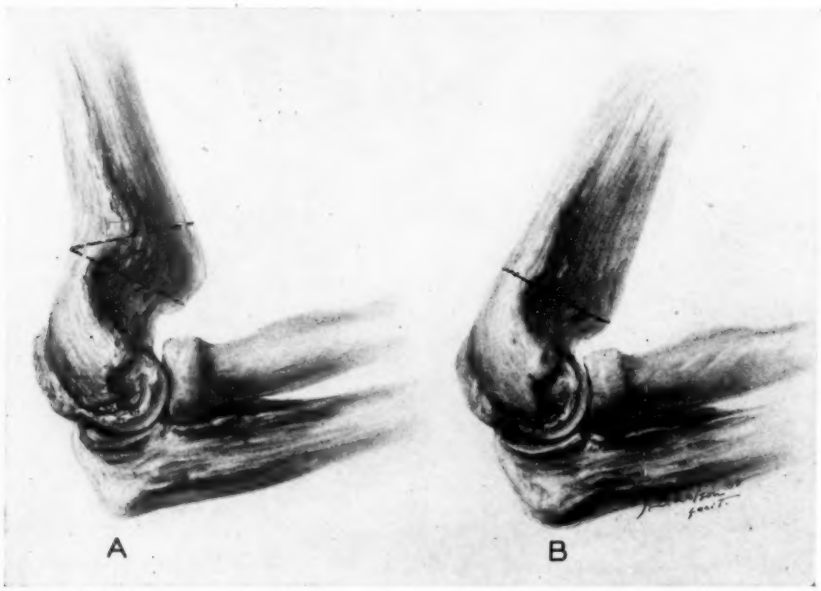

Fig. 7. Old supracondylar fracture with anterior angulation; A, note anterior bowing with position of wedge osteotomy; B, after osteotomy showing correction of angulation.

ing does occur it may be necessary to decrease the amount of flexion, and thereby the position of the fragments may be lost.

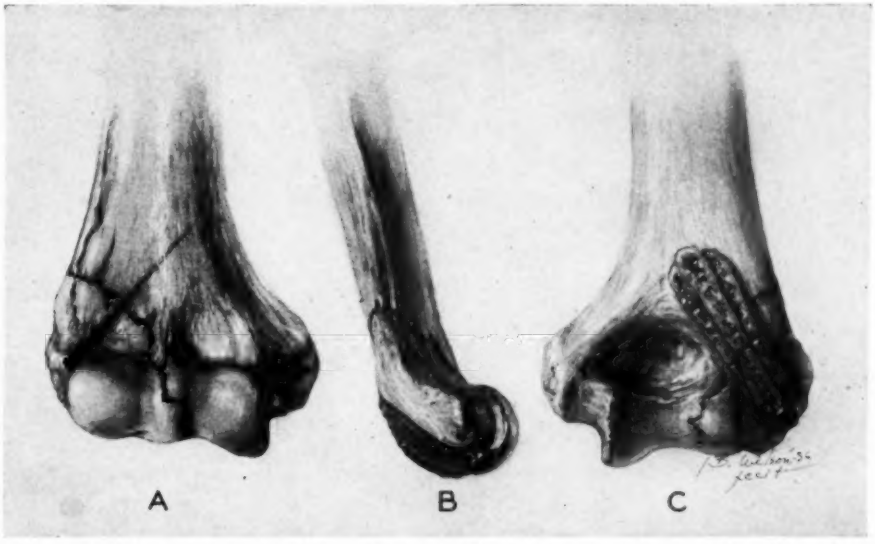

Fig. 8. Old fracture of external condyle; A, after operation; note small nail used to hold fragment in place; B and C, lateral and posterior views showing osteoperiosteal grafts. (*After Campbell, Speed and Macy.*)

Osteotomy for malunited supracondylar fracture may be done through a lateral or medial incision, the choice depending upon the type of fracture and the character of the displacement. If the osteotomies are done with careful dissection and minimal trauma the results are almost uniformly satisfactory. In all osteotomies about the elbow joint care should be taken to isolate the adjacent nerves and protect these from injury. It is often necessary to carry out transposition of the ulnar nerve from the posterior to the anterior aspect of the elbow.

Fractures of the External Condyle

Fracture of the external epicondyle is observed rarely. Occasionally the detached epicondyle fails to unite; when non-union is associated with pain the fragment should be excised. Fractures involving the anterior articular surface of the capitellum will be discussed as a separate group.

The external condyle, unlike the internal condyle, does not function in flexion and extension of the elbow joint and hence is not essential to elbow motion. Fracture of the external condyle may be followed, however, by the formation of excessive callus and an abnormal amount of fibrous tissue which limits the anteroposterior mobility of the joint. Most of the late fracture deformities in this area consist of detached external condyles which are adherent to the shaft of the humerus by fibrous tissue. The non-union is due to displacement and rotation of the condylar fragment. It has been said that displaced fractures of the external condyle should always be reduced by open operation. Only occasionally in the presence of displacement is it possible to secure by closed manipulation a reapproximation sufficiently accurate to insure a good result. In children, owing to the large amount of cartilage covering the bone, it is impossible to tell from the roentgenogram the extent of the fracture and the amount of the displacement. In the open reduction of this fresh fracture the condylar fragment should be accurately replaced and, if necessary, maintained in the corrected position by the use of small nails or autogenous or beef bone pegs. Any soft tissue attachment which may be present should be preserved, in order to insure a blood supply to the condylar fragment, although at times the preserva-

tion of a soft tissue connection is impossible. Occasionally fixation is secured by means of catgut sutures.

face of the humerus improved, and most or all of the valgus deformity corrected.

In adult cases it is often best to excise

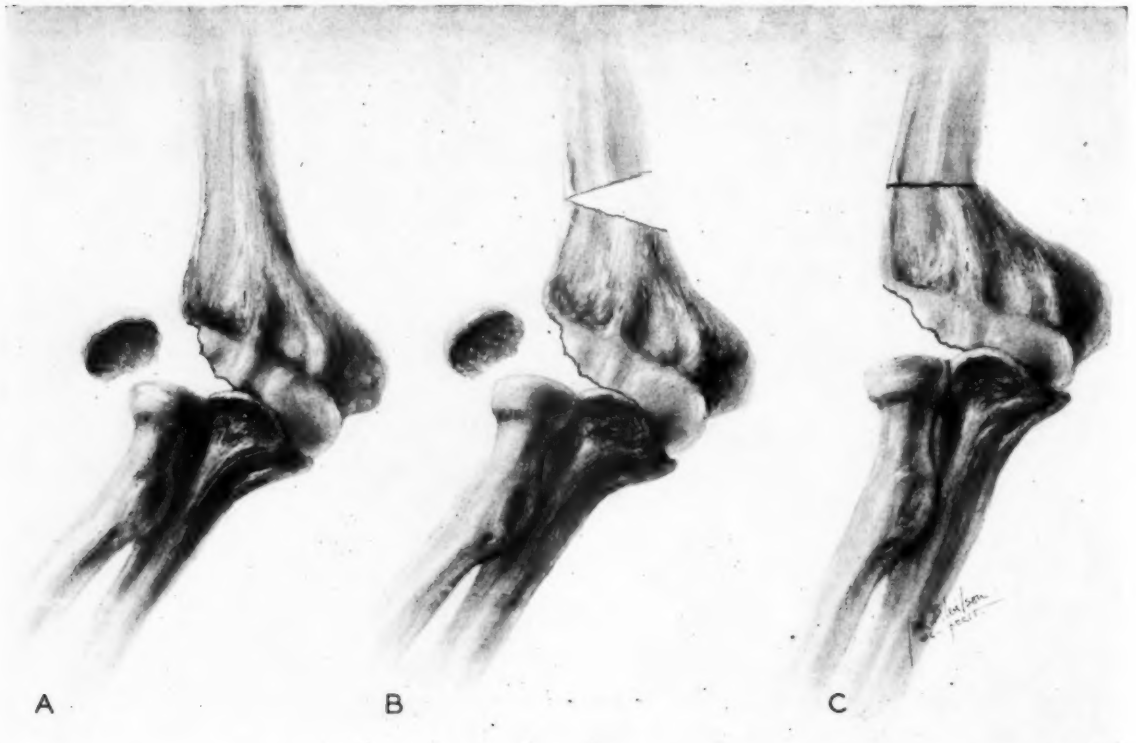

Fig. 9. Old fracture of external condyle of humerus with detached fragment and cubitus valgus deformity; A, before operation; B, showing position of wedge osteotomy; C, after operation; note correction of valgus deformity and removal of detached external condyle.

In old malunited fractures of the external condyle, occasionally it is possible and advisable to replace the condylar fragment. Sufficient bone should be removed from the fragments to secure freely bleeding surfaces, and either a nail or a peg should then be used to hold the fragments in place. Campbell, and Speed and Macey have recommended that osteoperiosteal grafts be placed over the posterolateral surface of the fragments after preparation of a suitable bed of raw bone (Fig. 8).

When valgus deformity is associated with ununited fracture of the external condyle, it may be advisable to perform a wedge osteotomy of the lower portion of the shaft of the humerus, the base of the wedge pointing medially (Fig. 9). In this way the level of the internal condyle is raised, the position of the articular surthe fractured external condyle and to make no attempt to replace it in normal position. Since many of the old cubitus valgus cases develop irritation of the ulnar nerve (discussed further under "Complications"), it is often advisable to follow excision of the condylar fragments by an anterior transposition of the ulnar nerve.

Fracture of the external condyle in a growing child, unless reapproximation is effected promptly, invariably results in cubitus valgus which becomes more marked as the child grows older. In such cases a wedge osteotomy is often advisable, the base of the wedge being placed well above the epiphyseal line on the medial side. In children removal of the external condyle is followed by a progressively increasing valgus deformity due to continued growth of the epiphysis of the internal condyle.

A series of cases in children reported by Speed and Macey shows the final results of replacing the external condyle several months after fracture to be very disappointing. Growth changes occur which lead to deformity and impaired function. When nailing of the external condyle is necessary in children, care should be taken to avoid putting the nail through the epiphyseal line. Bouman states that in fracture of the external condyle or capitellum the fragment or fragments should be removed, because of the possibility that they or the callus which forms about them may restrict the range of motion of the elbow.

Ashhurst reports the case of a boy of five years who was first examined five months after sustaining a fracture of the external condyle. A cubitus varus deformity, which is unusual in this type of injury, had developed. At operation the external condyle was found to be both rotated and displaced downward and backward, and a sufficient portion of the condyle was removed to permit extension and to overcome the varus deformity. The olecranon fossa was deepened to allow full extension, and the raw lateral surface of the external condyle was covered with fatty tissue. Three and a half years later, when the patient was eight and a half years old, there was no varus deformity and the range of extension and flexion was normal. Ashhurst also reports the case of a girl, aged nine years, who had had a fracture of the external condyle two years previously, resulting in complete detachment and anterior and outward rotation of the condylar fragment. Slight valgus deformity was present at operation. The condyle was chiseled to healthy bone and affixed with chromic catgut with the arm in full extension and supination. Two and a half years later firm union had occurred and there was a normal carrying angle and a normal range of joint motion. These cases are unusual but certainly show that excellent results can be obtained with the proper operative procedures.

Key and Conwell state that in adults it is always best to remove the external condyle, since owing to degeneration of the cartilage it is no longer suitable for function as an articular surface. In children they recommend always an attempt at replacement of the external condyle, because of the fact that the growth changes which follow its excision may lead to marked deformity.

FRACTURE OF THE INTERNAL CONDYLE

Because of its rarity, fracture of the internal condyle does not constitute so important a problem as the supracondylar or the external condylar fracture. The internal epicondyle or epitrochlea is fractured more often than the condyle or the trochlear surface. The epicondyle frequently remains unattached and later must be removed if limitation of motion and pain develop. Because of the articulation of the trochlea with the coronoid fossa of the ulna, fractures of the internal condyle are much more likely to be followed by limitation of motion that are fractures of the external condyle. Restriction of motion is particularly likely to result when there is much displacement of the internal condyle. Malunited fracture of the internal condyle usually leads to ankylosis, and arthroplasty is necessary before motion can be obtained.

After fracture of the internal condyle, bone may form within the elbow joint and cause locking. Excess bone formation is a very common sequel. Extension of the elbow is often restricted by the filling up of the olecranon fossa by callus.

Key and Conwell state that accurate reposition of the internal condyle should be carried out because it is essential to a good functional result. If the ulna is articulating in any way with the displaced internal condyle, however, it may be wise to leave the position of the condyle unchanged and to restore the alinement by an osteotomy of the humeral shaft. In such cases it is sometimes advisable to remove the normal external condyle since

it is not required for flexion or extension and is not essential to rotation of the forearm.

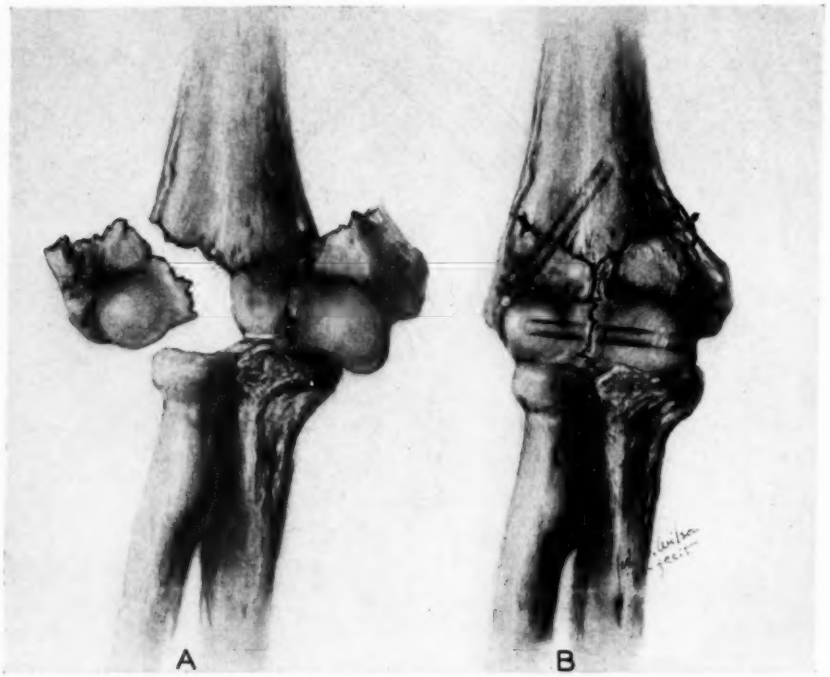

Fig. 10. Intercondylar T fracture of lower end of humerus; A, note wide separation of fragments; B, after open reduction showing beef bone pegs and wire. (*After Kellog Speed.*)

INTERCONDYLAR FRACTURES

This group includes all fractures which involve both condyles, of which the most important are comminuted "T" and "Y" types. This group undoubtedly includes most of the fractures which result in marked disability of the elbow.

When it is possible to reapproximate the condyles and reconstruct the joint, this should be done. Kellogg Speed reports several satisfactory results with the following technique (Fig. 10): an incision is made on each side of the joint above the condyles; the external condyle is freed and fastened to the shaft by an ivory peg or nail and the internal condyle may be fixed to the shaft by wire. In addition it may be necessary to wire or nail both condyles together. Accurate approximation minimizes the danger of subsequent bony ankylosis.

Campbell has recommended a similar type of reconstruction with the use of autogenous bone pegs. The condyles are first fixed to each other; another peg is then put through one condyle and up into the shaft of the humerus. When the condyles are extensively comminuted, the major fragments should be fixed in as nearly a normal relationship as can be secured. At times it is necessary to remove some of the smaller fragments. It is sometimes impossible to approximate the fragments accurately enough to create a new joint; in such cases resection is justifiable.

In occasional malunited intercondylar fractures it is only necessary to replace a single fragment: this is usually the internal condyle. If there is much deformity of the arm it may be necessary also to carry out an osteotomy.

A great many intercondylar fractures lead to complete or almost complete ankylosis of the joint. MacAusland has said that arthroplasty is indicated when the range of motion in such joints is 10 degrees or less. Key and Conwell state that an arthroplasty is nearly always necessary when there has been severe comminution

with the development of deformity and ankylosis.

Campbell states that in severely comminuted elbow fractures arthroplasty is indicated when the joint is destroyed so extensively that other operations are unsuitable, and when ankylosis has taken place without loss of the contour of the condyles. He further states that arthroplasty is seldom advisable in children. Occasionally, however, a child whose elbow has become ankylosed in full extension or in a position of disabling angulation is best treated by an arthroplasty of a modified type.

It is nearly always possible to improve both the function and the appearance of these joints if the procedure is chosen with judgment and the operation is effected without excessive traumatization.

FRACTURES OF THE ARTICULAR SURFACES OF THE CAPITELLUM AND TROCHLEA

This type of fracture is rare. Anatomically the capitellum and the trochlea are parts, respectively, of the internal and external condyles. Often only the anterior joint surface is fractured, especially in the case of the capitellum. Such a fracture is spoken of as "fractura rotuli humeri." The small fracture fragment is always displaced into the joint space (Fig. 11). Lindem, after reviewing the literature, has enumerated the following five clinical evidences of these fractures: (1) spontaneous pain in the region of the fracture; (2) normal elbow landmarks; (3) progressive limitation of flexion-extension motion to approximately 15 degrees; (4) a palpable bony projection in the antecubital fossa; and (5) crepitus caused by a loose body within the joint.

The treatment of the old fracture is removal of the displaced articular fragment. Excision should be done in the fresh fractures as well. Occasionally in young children, however, the fractured epiphysis of the capitellum can be replaced and fixed with chromic catgut sutures. Wilson reports an ununited fracture of the capitellum in which the fragment was replaced and fixed with a small bone graft; this resulted in an improved joint. Such cases, however, are unusual.

THE ANATOMIC VERSUS THE FUNCTIONAL RESULT

Joint structures are of little use if function is lost. The usefulness of joints is in direct proportion to their range of painless motion. Anatomic reposition of fragment after a fracture is to be desired, but the attempt to obtain this result should not be carried to the point of impairing or in any way risking the functional recovery.

This is especially true in children during the periods of active growth. Wilson has said that in children bony consolidation after fracture about the elbow always takes place; even when in the beginning there is an ugly deformity which seems to block the flexion of the elbow, the restriction of motion is eliminated in the course of time as growth at the lower epiphysis elongates the humerus and pushes the elbow away from proximity to the point of injury.

The loss or increase of the carrying angle is of little significance if function be preserved. Parents are most anxious, especially when the patient is a girl, to obtain recovery without deformity. It is much better, however, for the child to grow up with the ability to use the elbow normally than with a joint of normal appearance but limited use.

Cotton has stated that after fracture about the elbow in children he has rarely found ankylosis or any extensive loss of motion. He further states that even astonishing degrees of deformity due to inaccurate reposition may be followed in six months by good function. This is occasionally the case in adults. Often after intercondylar fracture the elbow remains broadened but its function becomes normal.

In cubitus valgus limitation of motion is rare. In cubitus varus motion is nearly always restricted.

König has shown that in children even marked deformities disappear after several years of absorption of bone and the filling

Fig. 11. Fracture of capitellum with anterior displacement.

up of its defects, and that only rarely does limitation of function result.

Ashhurst feels that when the elbow possesses a range of motion of from 50 to 150 degrees there is little disability. He also states that deformity alone is rarely a cause of disability. Ashhurst also calls attention to the fact that marked degrees of varus deformity usually weaken the elbow enough to impair its function, whereas slight degrees of cubitus varus are inconspicuous and cause no disability.

Gilcreest writes, "As structure is subservient to function, it is only natural to presume that the maximum return of usefulness in a limb would be brought about by the exact reconstruction of the deformed bone, but as perfect reduction is not infrequently impossible or inexpedient, our chief aim should always be complete restoration of function."

The clinical evidence and the opinions reviewed emphasize the fact that a good functional result is preferable to anatomic reposition of the fragments when such reposition necessarily entails impairment of function. Results in the treatment of fractures in the region of the elbow would be better if there were a more widespread appreciation of these fundamental principles of their care.

COMPLICATIONS

Complications of fractures of the lower end of the humerus are observed oftener in association with malunited fractures than with fresh fractures. Nerve injuries, Volkmann's ischemic paralysis, and traumatic myositis ossificans are the three most frequently observed complications. Excessive callous formation is occasionally a serious complication. Fortunately these complications are all uncommon and are observed in only a very small percentage of the total number of fractures about the elbow. In the present discussion space permits only a brief description of these complications and their symptomatology and treatment.

The *nerve injuries* probably form the largest group of complications. The ulnar nerve is involved more frequently than the median or radial. The ulnar nerve may be injured as a result of trauma at the time of the fracture, the lesion consisting of a partial or complete rupture; this occurs most commonly in association with fracture of the internal condyle. The nerve may be constricted by exuberant callus, or it may be traumatized gradually as the late result of a fracture of the external condyle which has been followed by the development of a valgus deformity: this results in delayed ulnar nerve palsy. Irritation of the nerve is due to the constant stretching and friction which it undergoes in the ulnar groove at the distorted elbow; often a small bulbous swelling of the nerve trunk appears at the point of irritation. The treatment of ulnar nerve injuries is the removal, when possible, of the causative factor. If a complete tear or rupture is suspected, the nerve should be explored and an attempt made to reapproximate its ends. If the nerve is caught in callus in or about the ulnar groove, it should be freed and transplanted to an anterior position, where it should be covered by fat and fascia. In delayed ulnar nerve palsy also, the nerve should be transplanted anteriorly. Any bulbous thickening of the nerve trunk

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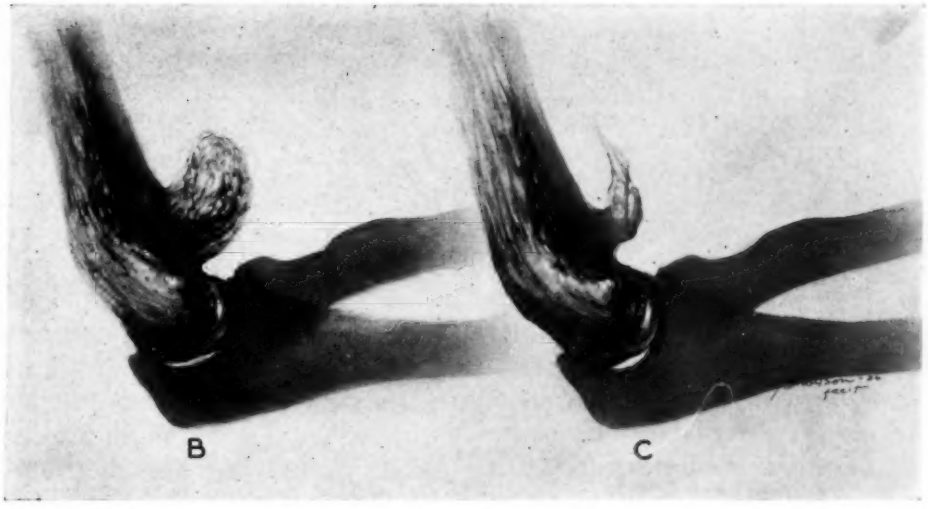

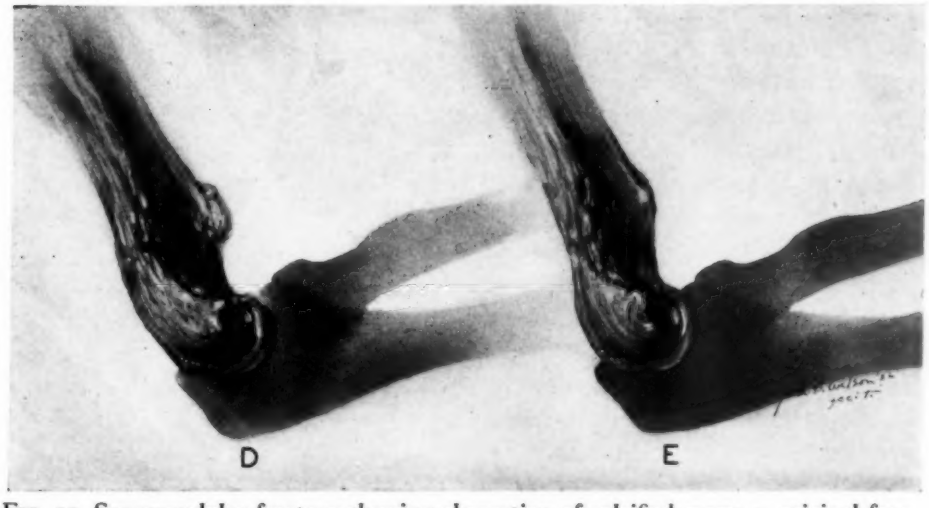

FIG. 12. Supracondylar fracture showing absorption of calcified mass; A, original fracture (*after Bristow*); B, three weeks after fracture; C, two months after fracture (*after Bristow*); D, four months after fracture; E, nine months after fracture with almost complete disappearance of calcified mass with rest. (*After Bristow.*)

should be excised, and the cut nerve ends should be reapproximated.

The median and radial nerves are seldom injured in fractures of the lower end of the humerus while the radial nerve is often injured in fractures of the head and neck of the radius. In most of these nerve injuries exploration is indicated; transposition is carried out if it appears advisable to remove the nerve from some projecting bone fragment or exuberant callous formation.

The *Volkmann's ischemic palsies* are undoubtedly the most serious complications of elbow fractures. They develop with extreme rapidity, resulting presumably from interference with the venous return from the forearm. Rapid degeneration of the fibers of the flexor muscles of the forearm is followed by fibrosis and contracture. These ischemic palsies may be caused by too tight bandaging but occasionally have been observed when no splint or dressing has been applied. In such cases the palsy results from the extravasation of blood into the antecubital space, where it is retained within a closed compartment formed by the fascial structures. The resulting distention causes compression of the antecubital blood vessels. When circulatory disturbances in the forearm and hand indicate that such changes are taking place, immediate relief of the pressure should be accomplished by operative evacuation of the extravasated blood. This is often the means of saving an arm or hand. When ischemic palsy has developed, treatment is at best unsatisfactory.

Traumatic myositis ossificans occurs most commonly in association with old posterior dislocations of the elbow. Occasionally, however, it is found as a complication of old fractures of the lower end of the humerus. Unmistakable limitation of motion is always present. Calcification is usually found within the brachialis anticus muscle; it may occur, however, in any place in or about the elbow joint. Myositis ossificans often follows a tear or stripping of the periosteum. It is impossible to foretell its development. After the presence of myositis ossificans has been proved by roentgenographic examination and sometimes by palpation, the elbow should be immobilized for a period of weeks or months. This is the best means of insuring absorption of the calcification. If the absorption does not take place after prolonged immobilization, it is advisable to explore and remove the excess bone in order to obtain increased motion and function in the joint. It is most inadvisable to attempt to manipulate or force motion in the presence of myositis ossificans, since manipulation usually increases the amount of calcification and results ultimately in more harm than good (Fig. 12).

SUMMARY

1. The commonly used methods of operative treatment of malunited fractures of the lower end of the humerus have been reviewed.

2. Emphasis has been placed upon the importance of a good functional result in preference to perfect anatomical reposition with questionable improvement of function.

3. A few of the common complications of malunited fractures of the lower end of the humerus have been discussed briefly.

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PULMONARY EMBOLISM*

GENERAL SURGICAL ASPECTS AND MEASURES FOR PREVENTION BASED ON A REVIEW OF THE LITERATURE

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LITTLE has been added to the symptoms and genesis of pulmonary embolism as described by the surgical pathologists, Virchow and Welch. Recent literature has dealt with the statistical data revealing the frequency of occurrence; while only in the past few years have preventive, prophylactic measures been advocated, notably by Walters and Bancroft.

Aschoff's[2] view on the origin of embolism and thrombosis are accepted as authoritative. He believes that the etiological factors are complex, and that no single factor is responsible for its formation. The conditions which precede thrombosis he catalogues as: (1) changes in the blood plasma; (2) changes in the blood elements; (3) changes in the blood flow; (4) changes in the vessel wall itself.

The cause of the high incidence of vascular thrombosis leading to pulmonary embolism in surgical cases as compared with medical cases is still obscure. While the medical cases occur in patients with debilitated or failing hearts—the surgical cases are usually observed in young or middle aged with normal hearts. The implication is—that the surgical procedures per se, or their sequelae, are responsible for the occurrence of pulmonary embolism (Allen).[1]

STATISTICAL ANALYSIS

Analytical reports from surgical clinics reveal the following interesting facts:

Incidence. Henderson[13] reported the incidence of fatal pulmonary embolism among surgical cases that came to necropsy at the Mayo clinic during 1917–1927 as 6 per cent.

There has been a definite and progressive increase in the incidence of thrombosis and embolism in the past decade. Nordmann[21] reports that in 1924 the incidence increased from 2 to 3 per cent, and in 1927 to 4.5 per cent. In the Giessen Clinic, Glahn[11] reports that a most marked rise began in 1925, during which the mortality from fatal cases of embolism increased 10 per cent. Bodon[5] states that according to his statistics the incidence has been four times as high since the war than in the period from 1911 to 1913. Kuhn, quoted by Walters,[30] reported the incidence of fatal embolism in Germany from 1924 to 1927 increased from 1.3 to 4.9 per cent, and that in 1927 thrombosis was found in every fourth necropsy, and fatal embolism in every twentieth body.

Age. Nicolaysen[21] reported that postoperative embolism is rarely found in patients under twenty years of age. The majority of fatal cases are well advanced in years, usually above the age of sixty. Killian, in a review of the Mayo Clinic statistics found the average age of patients affected to be higher than that of all other patients.

Sex. In the 51 cases of fatal pulmonary embolism in the Giessen clinic the ratio of males to females in Glahn's[12] statistics was 31:20.

Weight. Snell,[27] in a study of postoperative deaths of obese patients in the Mayo Clinic, found pulmonary embolism to be a more common cause of death than of the average patient. He states, "It suggests, but does not prove that obesity, per se, increases the liability of this com-

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plication. Difficulty of operation with the unusual trauma may be a factor; mild circulatory failure with resultant stasis might conceivably be more common in obese persons." Of the 84 patients who developed embolism in the Giessen Clinic, Glahn[12] reports 30 per cent were obese, 22.5 per cent were well nourished, and 41.5 per cent were cachectic.

Type of Operation. Walters[29] found that abdominal and pelvic surgery leads the list in about 70 per cent of cases. Stomach surgery, especially for cancer, and pelvic surgery, particularly operations for prostatic hypertrophy and fibroids of the uterus, are most important. Thyroid surgery is particularly free from postoperative embolism. Surgery of the extremities is rarely complicated by embolism. Henke called attention to the fact that oto-rhinological surgeons never see cases of acute fatal pulmonary embolism although they often see thrombosis formation in the great brain sinuses. After craniotomy acute embolism is very rare.

Anesthesia. It was formerly believed that a general anesthesia, especially ether, was a factor in causing embolus, but embolism cases are as frequent following local and regional anesthesia as in general anesthesia.

Preoperative Condition. According to Walters,[30] evidence that the formation of thrombosis and embolism in debilitated patients is frequent, is found in the fact that the incidence of fatal pulmonary embolism was three times greater after cystotomy preliminary to prostatectomy on debilitated patients than after the same operation performed on patients in good condition.

Kazda and Strocher[14] in a study of the autopsy material of the Pathological Institute of the University of Vienna noted the presence of cardiac changes in more than one-half of the cases of fatal post-operative embolism. Involvment of the respiratory organs, liver, and biliary tract was also found in about 50 per cent. These authors conclude that the surgical procedure superimposed upon the primary disease and the associated anatomical changes in the internal organs favored a distant thrombosis.

Infection. In Freund's[10] opinion, the importance of infection in the development of thrombosis has been overestimated. This is indicated by the rarity of thrombosis in long continued suppurative conditions, and its occurrence following completely aseptic operations. Lockhart-Mummery[18] states that sepsis is not necessarily an important factor in thrombosis.

Thrombophlebitis. It is significant that throughout the literature, relatively few cases of fatal pulmonary embolism were seen associated with thrombophlebitis clinically recognized. The concensus is, that when the clinical phenomena of thrombophlebitis appears, a clot has been formed which is not friable, and only small emboli are thrown off. So that *fatal embolism* is rare, but *infarctions* are common.

Intravenous Fluids. Huebschmann, in discussing Nordmann's[21] study of 143 cases, warned against the indiscriminate use of intravenous injections. Laven stated that he had often known thrombosis of the basilic vein to develop after continuous intravenous infusion. On the other hand, Bancroft[3] states he has been unable to find any increase in clotting elements of the blood after intravenous glucose administration.

Drainage. When thrombosis occurs in cases in which drainage is established, it develops from two to four days later, and the course is less severe than in cases without drainage, state Miller and Roepke.[19]

Seasonal Variation. No relationship between the season of the year and the occurrence of thrombosis and embolism was noted by Glahn.[12]

Lung Involved. The ratio of the right lung involvement to the left lung is 4:3. Pulmonary embolism occurs particularly often in the right lower lobe, reported Miller and Roepke.[19]

Origin. Fifty per cent of postoperative emboli have their origin in the femoral or iliac veins. Farr and Spiegel[8] in 16 autopsied

cases found that thrombosis occurred in the femoral, internal iliac, or common iliac veins, and more frequently on the *left* side than the right.

Occurrence. Fatal pulmonary embolism usually occurs at the end of the second week following surgery.

While this data has shown us the types of operations and the types of patients which are most prone to result in embolism, they have not shed any light on the etiology or prophylaxis. In order to study the problem best, we can arbitrarily divide these causes into two groups: (1) those caused by *physical* factors, and (2) those caused by *biochemical* factors.

It is generally conceded that trauma, infection, slowing of the blood stream and dehydration, predispose towards thrombosis. There must, however, be another factor inherent in the individual of a *biochemical* nature, which tends to make that individual more susceptible to thrombosis and embolism.

Let us discuss the physical factors which are concerned in the etiology and practical measures which have been used in the treatment of operative cases to reduce the incidence of embolism. Also, to discuss the studies made on the clotting factors of the blood, and to suggest certain lines of treatment which have been found to diminish clotting tendencies after operation.

The outstanding single physical factor is the slowing of the blood stream, with stasis of blood in the pelvic veins. Almost all pathologists and surgeons agree that slowing of the blood stream is one of the primary factors in the production of thrombosis.

It has been shown that thrombosis rarely occurs in arteries because the blood flow is too rapid. Fifty per cent of fatal postoperative emboli have their origin in the femoral or pelvic veins. While occasionally thrombosis may occur because of direct trauma, or from mass ligation, however, in most instances these veins are not in the immediate field of operation. The underlying cause is the slowing of the circulation in the veins. The factors which may slow the blood stream in the large veins occur commonly in surgical patients. Among the most important are: (1) loss of blood; (2) shock; (3) loss of fluids—with preoperative purgation, postoperative fluid restriction, increased sweating, and vomiting, there is a marked increase of fluid output over intake, with a resultant increased viscosity of the blood; (4) weak heart action following surgery; and (5) interference with the action of the respiratory movements due to tight bandaging and splinting of the abdominal muscles.

Any method which will cause an increase in metabolism, rate of blood flow or blood pressure, should decrease the incidence of embolism. Thyroid effectively increases metabolism. Plummer's observation that in hyperthyroid patients thrombosis and embolism seldom occur, even when disturbances in blood flow due to the associated cardiac decompensation is present, led Walters[29] to institute a regime in which dessicated thyroid, grains 2, three times a day, is given postoperatively, with a marked reduction in the incidence in the 4500 cases done on his service.

In agreement with Walters, Freund[9] markedly decreased the incidence of embolism by thyroid administration. He reports one case of fatal embolism in 4000 operations. Even in prostatectomies, he has reduced the incidence to 0.2 per cent. Freund warns against the simultaneous use of thyroid and insulin in diabetics.

EXPERIMENTAL EXTRACORPOREAL THROMBOSIS

Shionoya, Johnson and Rowntree[28] reported on their experiments in the production of thrombosis by the use of an extracorporeal cannula loop, the cannula being inserted into the carotid artery and jugular vein of rabbits. Mechanical, physical and chemical factors were varied to determine the effects on rate and type of thrombus formation. The marked influence of slowing of the circulation on thrombosis was quickly recognized. They found that anything that tended to retard the circulation would hasten the process of thrombosis. Conversely, when circulatory stimulants as thyroxin or ephedrin were injected into

the animal, the circulation was maintained considerably longer than normal without clotting occurring in the cannula.

If venous stasis is the outstanding single factor in the etiology, what then, may be done to influence the circulatory stasis? The following measures are at our disposal: (1) careful preoperative preparation, especially in those of advanced years, providing aid through digitalis to a weak or failing circulation; and (2) reduction of fluid loss by omitting preoperative purgation; by providing adequate fluid intake, and avoiding unnecessary inanition.

During Operation. Careful operative technique; clean cut dissections; accurate hemostasis; avoiding mass ligatures, and avoidance of undue pressure on great veins by retractors.

Postoperative. 1. Adequate fluid intake by intravenous, rectal or subcutaneous routes;

2. Intelligent use of circulatory stimulants;

3. Tight bandages should be avoided; especially should the free mobility of the chest be preserved. If we consider that the return flow of blood in the vena cava is largely due to the suction of the heart and the respiratory movements, then the increased abdominal pressure due to postoperative distention and the splinting of the diaphragm must cause considerable stasis in the veins of the pelvis. With the use of the Gatch bed and Fowler's position, we have the double factor of gravity and constriction in the region of Poupart's ligament increased by the flexion of the thighs and the lower border of tight dressings, to embarrass venous flow;

4. Existing preoperative varicosities should be bandaged;

5. Position of the patient should be frequently changed to overcome stasis;

6. In abdominal cases every effort should be made to prevent and reduce postoperative nausea, vomiting, and gastric dilatation by the early use of the Levin tube, and energetic measures instituted for combatting intestinal paresis in peritonitis cases. Bancroft[3] believes that distention is lessened if food is given early. Alvarez and others have shown that when a bolus of food passes through the pylorus, intestinal peristalsis is stimulated, which will carry with it gas as well as solid material. In uncomplicated cases, Bancroft routinely gives tea and toast the afternoon following the morning operation;

7. Routine employment of oxygen and CO_2 postoperatively for five minutes every two hours for the first twenty-four hours to prevent venous stasis by forcing deep respirations at a period when all vital functions are at low ebb;

8. Encouraging the use of early systematic exercises of the arms and legs, and especially daily elevation of the foot of the bed, as a mechanical method of increasing the venous flow from the dependent pelvic region. Pool,[24] in 1913, published an article on systematic exercise in postoperative cases in which he recommended exercises of the arms and legs be commenced the third day operative. Gamble[11] recently suggested using a pair of bicycle pedals mounted on a board base which can be placed conveniently for exercising the lower limbs.

Bettman of Leipzig, described a manual apparatus, a modified Perthe inflatable cuff which is attached to the bed, and controlled by a simple lever mechanism operated by raising and lowering the limb.

BIOCHEMICAL FACTORS

Evans[7] calls attention to the similarities between thrombosis following operation, parturition and the acute fevers. All of them follow tissue injuries with subsequent absorption of broken down products. In a study of the platelet count of postoperatives he found an increase from four to six days after operation, reaching its maximum about the tenth day, and then declining to normal during the subsequent ten days. He believed that the disintegration of the platelets provides material which is associated with the formation of thrombin, and the coagulation of fibrinogen.

Patey[23] argues that to prove the existence of a relationship between the numerical platelet increase and the liability

to postoperative thrombosis, it is necessary to prove that the operation of splenectomy is more liable to be complicated by thrombosis and embolism than other operations.

Koenig[15] believes that the vessel wall is injured by a toxin found in disintegrated muscle. He calls attention to the fact that thrombosis is most frequent in conditions accompanied by pronounced nuclear destruction, and in cases of tumor, inflammation and fracture. He believes detoxification can be accomplished by autogenous blood injections.

Allen[1] observed the blood changes following operation, and reported that fibrinogen is probably the most important of these changes. The number of platelets, cholesterol and bleeding time, showed no definite changes. The coagulation time and blood calcium showed slight variations. The number of erythrocytes and leucocytes, the prothrombin time, the fibrinogen and the lipoids showed definite and constant changes. The increase of leucocytes is significant because leucocytes are known to furnish thromboplastic substances that play an important part in coagulation.

Allen states "This non-specific physiologic change may partially explain the relatively high incidence of pulmonary embolism occurring in surgical patients as compared with non-surgical patients."

Bancroft and Stanley-Brown[3] performed experiments on dogs wherein vessels were ligated, causing necrosis of the organs they supply, such as the gall bladder and appendix and found an increase in the clotting factors of the blood.

Physiochemical studies made by Bancroft[4] and his associates over a period of years, have shown that patients with postoperative thrombosis and embolism have an increase of blood clotting elements in their blood. In studying the individual factors in blood clotting they found the following:

1. *Fibrinogen.* Experimental as well as clinical evidence seems to indicate that the concentration of fibrinogen within wide limits has little influence on clotting time. But inasmuch as any condition which brings about a concentration of the blood (such as dehydration) may show an abnormally high fibrinogen, and, as such blood has a high viscosity and is therefore subject to venous stasis, a condition favoring clotting . . . a high fibrinogen may indirectly indicate the possibility of danger from thrombosis.

2. *Calcium.* The independability of calcium in the clotting process has led to the belief that deficiencies or excesses of calcium must influence clotting. Clinically, normal blood calcium is found in hemophilia, and in the hemorrhagic diathesis of obstructive jaundice. In fact, there are no marked changes in the coagulation time in either the most marked hypocalcemia of parathyroid previa, or in the high blood calcium of hyperparathyroidism. No elevation in blood calcium has been demonstrated on spontaneous thrombosis.

3. *Thromboplastin.* This substance possesses the power of converting prothrombin into thrombin, in the presence of calcium. Unlike prothrombin, which appears to be limited to blood plasma, thromboplastin is widely distributed in the body. While platelets constitute the most important source of thromboplastin, it is probable that all cellular structures contain this substance, and liberate it when injured.

As the rate of clotting is dependent on the thrombin concentration in the blood it was their basis for developing a practical quantitative method of determining blood coagulation.

They[4] describe the technique of the plasma clotting test, which is a measure of the concentration of prothrombin and thromboplastin in the blood. They consider that 1.45 is the normal plasma clotting time. Using this time in seconds as the numerator, and the patient's plasma clotting time in seconds as the denominator, they obtain an index which they call the "plasma clotting index." This index normally ranges from 0.8 to 1.95. Readings below 0.8 are on the bleeding side, and those above 1.05 are on the clotting side.

They then attempted to determine whether the clotting factors could be changed by some therapeutic means. At the suggestion of Loeb of Columbia University, they used 10 c.c. of a 10 per cent solution of sodium thiosulphate intravenously, for three successive days. They

believe that sodium thiosulphate should be used prophylactically in patients having a high clotting index, and that it shortens the convalescence in cases of phlebitis. Its therapeutic value is prophylactic, rather than curative.

Mills[20] and Kugelmass[16] have shown that a carbohydrate and fat diet will raise the basal metabolism but will not increase blood clotting elements, while a protein diet not only raises metabolism but definitely increases blood clotting elements.

It is Bancroft's belief that in a number of cases, thrombosis and embolism may be aborted by administering a diet low in fats and carbohydrates, and the intravenous administration of sodium thiosulphate. In 1932 they reported 4250 operative cases treated under their regime with 11 cases of phlebitis, and no death from embolism.

CONCLUSION

In clinics where a definite prophylactic regime has been instituted both preoperatively and postoperatively, and drugs given to influence clotting factors, statistics show a marked decrease in the incidence of thrombosis.

SUMMARY

Until more definite facts are known concerning the chemistry of blood coagulation and the therapeutic control of clotting factors, our efforts must be aimed at the removal of factors which predispose to thrombosis, and most especially venous stasis in the large veins of the pelvis.

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CASE REPORTS

PREGNANCY IN RUDIMENTARY HORN OF BICORNUATE UTERUS

SPONTANEOUS RUPTURE; OPERATION WITH RECOVERY

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PREGNANCY in the rudimentary horn of a bicornuate uterus although not one of the rarest happenings in obstetrics is by no means common. Such a case is always of interest because of the drama associated with it and the satisfaction resulting from a successful termination.

Many women with congenital abnormalities become pregnant and abort with no complications. Often the abnormality is not recognized even then. It is a not unusual occurrence for women with these defects to go to term and have normal spontaneous deliveries.[2–5] Frequently, then too, the abnormality goes unrecognized. Thus Zanela[1] in reporting 6 cases of ruptured uterus mentions one woman who delivered her fourth child before the uterus ruptured. Another patient in this series ruptured her uterus in her fourth pregnancy with a transverse presentation. Each of these women had a bicornuate uterus with a single cervix.

The fact that one horn of such an abnormal uterus may have no connection with the other nor with the vagina is also mentioned in various textbooks.[2,5] Guerrant[6] reports from Ladin's clinic a primipara in whom rupture of a right rudimentary horn occurred when she was four months pregnant. She was operated upon and recovered. Fabris[7] reports a case of rupture in a rudimentary horn at three months. Sheldon,[8] reporting 26 cases of rupture of the uterus, describes in his Case XXI, a patient, para V, who, at six months gestation, ruptured the left horn of a bicornuate uterus and recovered. Gordon,[9] reports a case in which there was fairly wide separation of the rudimentary horn from the larger one. This patient had rupture occur at the fifth month in her first pregnancy.

Although one usually associates rupture of the uterus with a patient critically ill, Kelly and Noble[10] state that " . . . in some cases of spontaneous rupture during pregnancy, the haemorrhage is so slight as to pass unobserved and the accident is discovered only when operative procedures become necessary for removal of the fetus."

The case reported here adds to the literature another case of pregnancy in a rudimentary horn of a bicornuate uterus in which external migration of the sperm occurred. This case terminated in spontaneous rupture, at five and one-half months followed by operation and recovery.

R. D., aged twenty-four years, was admitted to the Greenpoint Hospital, November 5, 1936. She had been married for a year and a half and had had her last menstrual period on April 18, 1936, five and a half months previous to admission. This was her first pregnancy.

The pregnancy had been normal except for moderate pain in the lower abdomen in August and October, 1936. This pain had been sharp enough to limit the patient's activity but not to incapacitate her. At four o'clock on the morning of admission, the patient experienced severe

pain in the epigastrium and in the region of the umbilicus. A physician was called but could feel no labor pains. He listened for and heard the fetal heart and administered morphine sulphate grain ¼ to relieve the pain, following which the patient vomited. At nine A.M. a second, more severe attack occurred, accompanied by vertigo and faintness.

Fig. 1. Schematic representation of findings at operation.

On admission to the hospital at eleven A.M., the following observations were made.

"The patient is a well nourished, well developed young adult female, acutely ill. She is in shock. Her pulse is 140 beats per minute, her blood pressure is 48/25. The skin is pale, lips cyanotic. There is a cold perspiration on her face. The heart tones are distant. The abdomen is distended, a swelling reaching from the symphysis to a point halfway between the navel and the ensiform. No fetal movement is visible nor palpable. The fetal heart is not audible. The fetal parts are easily palpable; no uterine contractions are made out. In either lower quadrant, there is exquisite tenderness with marked rebound tenderness. No masses are felt but there is marked tenderness in either vaginal vault."

Fig. 2. *X*-ray picture following lipiodal injection.

Morphine sulphate grain ¼ was administered and a solution of 5 per cent glucose was given by hypodermoclysis. The patient was placed in Trendelenburg position. The blood pressure readings were taken at fifteen minute intervals; by five P.M. it had risen to 90/60, and the pulse rate was 130. Since there was no further improvement 500 c.c. of blood was given by indirect transfusion and the patient's abdomen was opened.

A large quantity of free blood and blood clots were found. The omentum reached into the pelvis. Behind it an amniotic sac containing a dead fetus (Fig. 1) was found. This sac was ruptured and the fetus was delivered. It was then discovered that the placenta was protruding from a rent in the posterior wall of the left horn of a bicornuate uterus. This uterus measured 10 cm. in diameter. Attached to it were the left tube and ovary. A very definite band of constriction separated this structure from the right horn. It was easy to apply two clamps across this band and remove the left horn.

Following operation, a second transfusion of 250 c.c. of blood was administered. Except for a moderate morbidity and some separation of the wound edges, the patient made an uneventful recovery and left the hospital on the eighteenth day after operation.

When the specimen was examined, one was unable to make out a canal at the site of transection. Numerous blocks were cut from this region for microscopic study but none of them revealed anything resembling an endometrium.

Two months after operation, the patient was seen again. The abdominal wound was healed. Lipiodol was injected into the cervix and an *x*-ray picture was taken which showed one uterus and one patent tube (Fig. 2). It failed to give any suggestion of a remnant of a left uterine horn.

DISCUSSION

Although the failure to find a canal at the site of transection of a rudimentary left cornu is not conclusive evidence of its absence, two other findings point to this conclusion. One is the fact that a band of tissue separated the left cornu from the rest of the uterus and the other, is the absence of any evidence of even a minute canal going toward the left side of the retained uterus in Figure 2. We must therefore deduce that we are dealing with another case of "wandering fertilized ovum"[11] or transperitoneal migration of the sperm.

SUMMARY

A case is reported of a twenty-four year old primigravida in whom spontaneous rupture of a rudimentary uterine horn occurred after five and one-half months amenorrhea. The ruptured horn and dead fetus were removed at laparotomy. The patient recovered. Two months later an *x*-ray picture taken following transuterine injection of lipiodol showed the remaining horn and tube of a bicornuate uterus.

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EPIGASTRIC HERNIA CAUSING SEVERE SYMPTOMS*

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TEXTBOOKS on surgery as a rule discuss epigastric hernia rather briefly and inadequately. They all state, however, that the condition is a cause of various symptoms which may be confused with those of intra-abdominal disease. From time to time, we need to be reminded of such obvious facts in cases seen rather infrequently by the general practitioner. This particular point was recently emphasized for me by the following case.

J. S., aged sixty years, male, came to the hospital April 11, 1936 complaining of severe abdominal pain.

Past History. When sixteen years old he was operated on for harelip. While taking the anesthetic he began to vomit and was struck across the abdomen by one of the doctors to start him breathing. Patient dates his stomach trouble of long standing from this occurrence. He began to have attacks of stomach ache after this, lasting for a day or two. These spells occurred from a week to a year apart, but gradually increased in frequency and severity. In the interim he suffered constantly from indigestion. He has had much treatment but no surgery. Eight years ago he spent three months in a hospital in Tacoma under treatment for ulcer with very little benefit. He had typhoid fever at twenty-four years, but no other serious illness. He has had no acute attacks of pain for three years preceeding the present illness but has been on a diet constantly for indigestion.

Present Illness. March 20 at 3 P.M. he suffered a severe abrupt attack of pain in the pit of the stomach. He had eaten a meal of meat loaf in a restaurant a half hour previously. This was a departure from his customary diet and was attributed by the patient as the cause of the attack. After the pain started he vomited and was partially relieved but the pain has continued intermittently since. He has vomited daily, sometimes five times. He has been on a milk diet.

Examination showed a robust well preserved male, 6 feet tall, weighing 190 lbs. evidently in extreme pain and appeared in mild shock. There is evidence of a repaired hare lip and complete cleft palate. His temperature was 100:4; the pulse rate 104, slightly irregular; blood pressure 110/70. The physical examination was negative except for the abdomen.

There was no distention or rigidity, except that pressure in the epigastrium provoked resistance. There was a palpable boggy mass in the lower left quadrant, probably feces and a 5 cm. soft tender, irreducible tumor just above the umbilicus. No opening in the facia could be felt.

The patient's appearance on admission was such that a ruptured peptic ulcer was considered, but as this did not square with the physical findings a policy of watchful waiting was followed, and the acute pain gradually subsided somewhat. The urine and blood were normal and the Kahn test was negative. *X*-ray examination showed normal contours with increased peristalsis.

On April 15, under avertin and procaine, an incision was made over the linea alba. The tumor proved to be a fatty mass larger than it appeared from palpation and flattened out about 7 cm. in diameter. There was a circular opening through the fascia about 2 cm. in diameter through which omentum protruded. The sac could not be distinguished except at the edges of the opening. Exploration of the abdomen revealed no pathology except that the vessels of the stomach appeared engorged. The mass, continuous with omentum was excised, the peritoneum adherent to the ring separated and closed, and the opening in the fascia overlapped laterally.

On the day following the operation the patient had a profuse bloody emesis and considerable pain for four days. Following this the convalescence was uneventful and he was dismissed on the eighteenth postoperative day. Up to this time he has been entirely relieved of his symptoms.

An epigastric hernia is formed, according to Quain[1] by a small tag of preperitoneal fat or the fat in the fold of the hepato-umbilical ligament forcing itself

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through the transversalis and rectus fascia at the site of the opening for a blood vessel. These most often occur in the linea alba. Most of these hernias remain quite small, are not hernias of the abdominal contents and do not involve the peritoneum. Small hernias elsewhere are often difficult to detect by palpation from without. Paradoxically the small epigastric hernias can not be detected by palpation from within. This point must be kept in mind in exploration. A certain percentage, not more than 1 in 20 according to Moschcowitz,[2] continue to enlarge until the opening in the fascia permits intra-abdominal pressure to force the peritoneum through it followed by abdominal contents, usually omentum.

One would naturally expect these larger hernias with formed sacs to cause the more severe symptoms. However, this does not appear to be the case. Quain[1] states that epigastric hernias with well formed sacs cause no more symptoms than smaller ones; Sullivan and Antupit,[3] that it is the small hernias the size of a pea, that cause the most symptoms of abdominal disease.

The symptoms and signs of epigastric hernia may quite properly be divided into two groups: (1) those local symptoms directly associated with the hernia, namely tumor, tenderness at the site of the opening, local and superficial, due to preperitoneal fat caught in the ring and often relieved by recumbency; and (2) symptoms not directly associated with the hernia and simulating such a variety of conditions as gastric cancer, peptic ulcer, gall-bladder disease, chronic appendicitis, and intercostal neuralgia.

While most surgeons seem to accept this latter class of symptoms as sometimes being caused by epigastric hernia the opinion is by no means unanimous. Lewisohn[4] does not believe that simple epigastric hernia causes serious gastrointestinal symptoms. He observed 13 cases over a period of three years in which the repair of such a hernia did not relieve symptoms. Eleven of these cases proved to have lesions in the stomach of duodenum. He also emphasizes the frequency of co-existing disease with epigastric hernia.

Moschcowitz[2] believes epigastric hernia is responsible for intra-abdominal symptoms in only a small percentage of cases; Gilbride,[5] that the hernia does not cause symptoms simulating intra-abdominal disease and that these are due to associated conditions.

On the other hand, Friedenwald and Morrison[6] in a comprehensive review report 65 cases causing symptoms in their experience. Eleven of these were confused with chronic appendicitis, 7 with gall-bladder disease, 7 with duodenal ulcer, and 3 with cancer of the stomach. Most of these cases were entirely relieved by repair of the hernia. They state that many patients who have been treated for years for ulcer, gastritis, nervous dyspepsia, and gallstones, are cured by repair of the hernia.

I believe that in the course of routine physical examinations these small hernias are discovered not at all infrequently and that they seldom cause any severe symptoms. With no records to support it, my personal belief is that it is only the hernia involving intra-abdominal contents that causes intra-abdominal symptoms.

SUMMARY

1. One case of epigastric hernia involving abdominal contents is reported which was apparently responsible for severe symptoms of intra-abdominal disease.

2. It is still a controversial matter whether or not simple epigastric hernia simulates intra-abdominal disease.

3. In exploratory procedures epigastric hernia should be kept in mind when intra-abdominal disease is expected, and vice versa; also the possibility of the two conditions co-existing. Further, the anatomical considerations making small hernia not evident from within the abdomen are to be considered.

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VOLVULUS OF RIGHT COLON AND MECKEL'S DIVERTICULUM*

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PHILADELPHIA

VOLVULUS of the right colon is less common than volvulus involving the left colon. This is due, of course, to the fixation of the right colon. There is almost invariably a congenital abnormality present in order that volvulus of the right colon can occur.

Insufficient intestinal fixation is by far the most frequent cause of obstructive symptoms due to anomalies of intestinal rotation and the most frequent form of obstruction is volvulus.

O. J., male, white, ten years old, was admitted to Medical Service B. Jefferson Hospital, November 18, 1929, complaining of pain across the abdomen and vomiting.

There were five brothers and sisters, all living and well.

Past History. He weighed four pounds at birth and was "difficult to raise." He had many illnesses. Five years ago, he had what was thought to be appendicitis, but was not operated upon. Every few months, for the last five years he has been subject to attacks of cramp-like and intermittent pain in the abdomen referred to the navel which lasted forty-eight hours, and was associated with vomiting every fifteen or twenty minutes. During the interval he was perfectly well. He has always been constipated and used laxatives or enemas continually.

Present Illness. At 2 A.M., November 17 he was awakened with severe cramp-like pains in the abdomen and vomiting. The vomiting continued until 6 A.M. Wretching continued thereafter until admission at 5 P.M. November 18. The symptoms subsided suddenly and he was perfectly well during the remainder of his stay in the hospital.

Laboratory Studies. Blood count, gastric analysis, gall-bladder drainage, stool and urine examinations were normal.

X-ray Studies. November 21, 1929. No evidence of organic disease. There was some gastric retention but no cause could be found. The cecum freely movable and was not tender.

Diagnosis. Gastrospasm. Cholecystogram and flat plate of abdomen negative.

Physical Examination. A well nourished young white boy, with no abnormal findings. The abdomen was flat; no masses were palpated but there was tenderness on deep pressure at the navel and in both flanks.

He was discharged November 27, 1929, with the diagnosis of gastrospasm.

August 24, 1931 he was readmitted with the same complaints and after two days in the hospital the father took the child home after refusing operation. It was thought that there was a Meckel's diverticulum present. No *x*-ray studies were made at this time.

He was again readmitted March 14, 1933. Since his release from the hospital August 26, 1931, he has had his usual attacks of pain and vomiting but has always been relieved in a few hours by rest in bed and hot water bottles. At 4 P.M., March 13, 1933, an attack began suddenly while at school. The vomiting continued for two hours. The pain increased in severity and about 7 P.M. the right side of the abdomen was becoming distended. He was driven forty miles in an automobile to the hospital.

Physical Examination. The patient was lying on his back with his knees drawn up and having spasmotic attacks of abdominal pain. The head and chest were negative.

The abdominal wall was tense, and the abdomen distended assymetrically, mostly in the right. There a cystic, tender mass occupied the whole right side of the abdomen and appeared to be distended bowel. Diagnosis of volvulus was made and the patient operated upon.

Operation. March 14, 1933, ether anesthesia.

* Read before the Philadelphia Academy of Surgery, December 2, 1935.

On opening the abdomen through a right paramedian incision, a large quantity of dark brown fluid escaped. A large, cystic, black

FIG. 1. Specimen removed at operation.

mass presented itself and was found to be cecum, ascending colon and ileum. There was a Meckel's diverticulum attached to the umbilicus by a cord. The cecum, ascending colon and distal ileum were rotated over from right to left making one complete turn. The whole mass then was found to be across the cord from Meckel's diverticulum. This cord was cut and the mass delivered. The vessels were thrombosed and the intestinal wall gangrenous. The ileum proximal to the diverticulum was cut across between Payr clamps. The cecum and ascending colon were attached by a very long mesentery and the axial center of rotation was the proximal transverse colon. The transverse colon proximal to the midcolic artery was cut between clamps and the mass removed. A side-to-side anastomosis was made between the ileum and transverse colon using the Kerr technique. The edges of the mesentery were brought together and the abdomen closed with a drain in the pelvis.

Progress. The patient was treated with intravenous glucose and saline by hypodermoclysis and duodenal tube. There was marked distention. On the seventh day he had several copious dark watery bowel movements. Thereafter his convalescence was uneventful but for a slight infection of his wound. He was discharged the thirty-fifth day as well.

A third readmission was on November 9, 1934. The patient had been perfectly well since the previous operation. He had had none of the digestive disturbances that he had previously and has not had to use laxatives. That day, after lunch he had a sharp pain in the abdomen with vomiting. The pain was spasmodic and severe. One enema was effectual at 2 P.M. but he had passed no gas or feces since. The pain has continued.

Examination. The abdomen was distended, tense and tender; peristalsis was hyperactive.

Diagnosis. Obstruction.

Operation. A low midline incision was made. Distended ileum was found in lower abdomen. A band of adhesions had anchored the ileum to part of the scar of the previously excised mesentery of the ascending colon. This was cut and ligated. There were no other adhesions in the abdomen. The site of the anastomosis done six months ago was satisfactory and the stoma was probably 1½ inches long. The abdomen was closed without drainage.

Recovery was uneventful.

He has been seen several times since his last operation and has had no further trouble.

Rotation of the intestinal tract occurs in three stages. The first step takes place between the fifth and tenth week while the loop of midgut lies in the umbilical cord. The second stage is characterized by the further elongation and finally a return to the abdominal cavity of the whole intestinal canal. Fixation is maintained by the vitelline duct and the vitelline artery which is the termination of the superior mesenteric artery. The cecum is caused to descend to the right loin by further elongation of the colon. This is complete about the eleventh week. The term "non-rotation" refers to a failure in the second stage. The third stage is characterized by the further descent of the cecum and fixation of certain portions of the intestine to the posterior abdominal wall by fusion of their mesenteries with the posterior parietal

peritoneum. The third stage is complete usually about the first month.

The predisposing causes of volvulus are (1) an unduly narrow loop or group of loops of intestine, (2) undue length of mesentery, and (3) a point of adhesion at the convexity of the loop, which can act as an axis of rotation. The exciting causes are (1) unusual effort or accidental movement of the body, (2) the peristaltic motility of the intestine, and (3) undue distention of the intestine. In this instance we have two factors to consider as the cause of volvulus; (1) an elongated mesentery or a failure fixation of the cecum and ascending colon and (2) fixation of the convexity of the loop by the remains of the vitelline duct. The volvulus occurred in this instance by a rotation of the cecum, ascending and proximal transverse colons and the terminal ileum on its mesenteric attachment. The mass then folded over the remains of the vitelline duct.

SUMMARY

A case of volvulus of Meckel's diverticulum is reported, illustrating the fact that gastrointestinal symptoms in growing children may be the result of certain congenital anomalies which may require rather radical surgery. Congenital anomalies of the right colon are probably more common than previously supposed. This patient had three anomalies which produced symptoms.

When it [fecal impaction] is situated beyond the reach of the finger, or if of too firm a consistency to be easily manipulated, inject 8 or 10 fluid ounces of warm mineral oil, olive oil, or cottonseed oil, with the patient in the knee-shoulder position. Allow this to remain for 12 hours, and it will so soften and disintegrate the mass that it can be usually passed without any difficulty.

From—"Synopsis of Ano-Rectal Diseases" by Louis J. Hirschman, M.D.

ACUTE REGIONAL (TERMINAL) ILEITIS

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TERMINAL ileitis is described as a chronic and nonspecific granulomatous disease. It has been suggested by Crohn that an acute stage exists. The purpose of this case report is to record an instance of acute terminal ileitis and to review the suggested forms of treatment.

Lee and Bockus,[1] state that it is possible that the chronic form of this disease may result from an acute stage, and refer to the case reported by DeCourcy[2] in which an operation upon a girl twenty years of age revealed marked inflammation of the terminal twelve inches of ileum. The inflamed bowel was returned to the abdomen and the patient made a complete recovery after a very stormy convalescence.

Mixter[3] quotes the grouping of regional ileitis suggested by Crohn:

Group 1. This group shows signs of acute intra-abdominal inflammation, indistinguishable preoperatively from acute appendicitis. There are pain and tenderness in the right lower quadrant, cramps, fever and leucocytosis. A mass may be palpable. At operation the terminal ileum is reddened, greatly thickened and bleeds readily, and its mesentery is edematous and contains enlarged hyperplastic lymph nodes. The appendix may be reddened and involved by contiguity but it shows no mucosal inflammation.

Group 2. The second stage presents symptoms suggestive of ulcerative colitis. There is colicky abdominal pain and looseness of the bowels, at times with blood and mucus. There is slight fever, malaise, marked loss of weight, and anemia, often severe, develops.

Group 3. The stenotic stage follows the ulcerative phase. Due to the extreme thickening of the bowel wall the lumen of the bowel becomes constricted and may be partially obliterated by the healing of the mucosal ulcerations. Usually the narrowing is most evident near the ileocecal valve, while above, in the more recently involved portion of the bowel, the earlier ulcerative phase predominates. The symptoms are those of partial obstruction of the small intestine. In this stage a mass is usually felt.

Group 4. The stage of fistula formation. This is a late manifestation of the disease and is the result of slow perforation of the ulcers. Frequently multiple fistulas are formed which may open externally through the abdominal wall or may be internal and at times can be demonstrated by roentgenologic examination of the gastrointestinal tract. In contradistinction to appendiceal fistulas which tend to heal, such fistulas are persistent and resist operative attempts at closure.

Mixter stated that

> they have operated upon only one case that might have been placed in the early phase of the process. In this case an immediate resection and anastomosis were performed with prompt recovery. The question may well be raised whether or not a procedure of this magnitude was indicated in the early stage of the disease and whether a temporary illeostomy or ileocolostomy might not have sufficed. In answer it can be said that the disease tends to progress, and where graded procedures have been employed we have generally found that the involved segment of bowel has later developed some form of complication, whether abscess or fistula, that has rendered resection much more difficult and hazardous.

In discussing Mixter's paper Dr. Emmet Rixford referred to an acute case that came

[1] LEE and BOCKUS. *Ann. Surg.*, 102: 412–421 (Sept.) 1935.

[2] DECOURCY. *Jour. Med.*, 15: 216, 1934.

[3] MIXTER, C. G. *Ann. Surg.*, 102: 674–694 (Oct.) 1935.

under his care eight years previous, in which the lower third of the ileum was swollen, red, and covered with an exudate. The appendix was removed but no operation was performed upon the ileum. Prompt recovery occurred with no recurring symptoms to date.

M. Q., a female aged eight years, was admitted to the Misericordia Hospital on March 17, 1936 with a temperature of 99 degrees, pulse rate 90, and respiratory rate 24. Urine examination was negative; hemoglobin 74 per cent, erythrocytes 4,370,000 and leucocytes 31,000.

The chief complaint was abdominal pain associated with vomiting. Four days prior to admission the patient developed headache, nausea and vomiting. On the day previous to admission abdominal pain developed which was most marked in the right lower quadrant.

General physical examination was negative except for rigidity over the lower abdomen, particularly on the right side. The father died of tuberculosis at thirty-three years of age, after having been confined to a sanitarium nine months prior to his death. There are four other children in the family, all well. The mother is in good health. With the exception of measles and whooping cough four years ago there are no other important items in the family history.

A diagnosis of acute appendicitis was made and operation was performed on the day of admission. Upon opening the peritoneal cavity an excessive amount of peritoneal fluid was present, and an extensive inflammatory process involving the terminal portion of the ileum. The bowel wall was covered with a thick layer of fibrin which was removed and sent to the laboratory for examination. Three small areas, measuring ¼ inch in diameter, were darker than the major portion of the bowel, and appeared to be early areas of gangrene. The thickness of the bowel wall was very definite and extended from the ileocecal valve for a distance of about 8 inches. The edema gradually subsided to normal bowel. There was no evidence whatever of any inflammatory reaction in the cecum. The mesenteric lymph nodes were enlarged but there was no pathology in the mesentery which would suggest mesenteric thrombosis. There was no evidence which might suggest that the pathology in the bowel was the result of a relieved intussusception. Cultures were made but no organisms were found. A first stage Mickulicz resection was performed and the abdominal cavity closed around the exteriorized bowel.

On the third postoperative day the areas of discoloration had increased in size and it was thought advisable to resect the loop of intestine. Blood culture was negative. The patient died on the fourth postoperative day.

Autopsy revealed that the patient died from a generalized peritonitis which was apparently primary inasmuch as there was no evidence of leakage at the operative site. The lungs showed evidence of a healed tuberculous nodule in the left lower lobe associated with pleural adhesions. The mesentery showed no evidence of vascular occlusion. The nodes were hyperplastic. The pathological report on the appendix was acute periappendicitis. Microscopic examination of the fibrin revealed nothing definite. A similar examination of the resected ileum revealed the following:

PATHOLOGICAL REPORT

Gross Description. Specimen consists of 12 inches of resected ileum of normal calibre. The wall is uniformly thickened and edematous except the proximal 2 cm. which is normal in thickness. The serosa is covered with a thin layer of fibrin and the bowel presents a dusky appearance with scattered irregular black areas. The mucosa is smooth, thickened, velvety; it presents no definite ulcerations but has a variegated color with scattered petechial and small gangrene areas. The bowel is cut close and no mesentery is submitted with the specimen. The blackish areas of gangrene are more numerous near the mesenteric attachment.

Microscopic examination of three sections from the thickened portion of resected ileum. The serous surface is covered with a thick layer of fibrin. All layers of the bowel wall are involved in an acute diffuse suppurative inflammatory process, are edematous, show scattered areas of hemorrhage and much cellular exudation with polymorphonuclear leucocytes predominating. The mucosa shows partial degeneration and there are no areas of ulceration.

Diagnosis. Acute diffuse suppurative inflammation of ileum.

COMMENT

At the time of operation it appeared that an extensive resection of the ileum would result in a fatal outcome. For this reason the cecum and inflamed portion of the ileum were brought out of the abdomen and the peritoneum carefully sutured around the bowel. The procedure was not justified by the result.

In personal communications from Dr. Mixter and Dr. Crohn they suggested that an ileocolostomy, combined with resection, would probably be the best procedure to follow in this disease. Although this extensive resection appears hazardous in the presence of such acute inflammation the recoveries reported justify the procedure. The case reports of DeCourcy and Rixford show that recovery will occur without resection. Only through accumulated statistics will it be possible to finally determine the best method to follow.

THE Preparation of Suitable Maggots. (a) The securing of a suitable variety of fly. (b) The constant production of large quantities of eggs. (c)The sterilization of the maggots.

The last is the crucial difficulty, and requires the activities of a skilled entomologist plus pathologist.

From—"Recent Advances in Orthopaedic Surgery" by B. H. Bruns, F.R.C.S. and J. H. Ellis, F.R.C.S.

STUDIES OF THE NATURAL HISTORY OF GENITOURINARY TUMORS. I: PRIMARY CANCER OF URETER

AUTOPSY STUDY WITH REVIEW OF THE LITERATURE

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THE purpose of this study is to add an autopsy report of primary cancer of the ureter to the 40 previously published cases. Twenty of these reports show that no operative intervention was attempted and that the majority of the others had exploratory operations only. From this data the natural history processes of this disease can be estimated. This study has been directed particularly to the duration of symptoms previous to observation and the total length of life following diagnosis or operation. Since only reports of unquestioned primary malignant tumors have been accepted a composite study of the metastases and their extension has also been made.

A full abstract of these accepted cases is not presented as recently several complete reviews have been published. Points of unusual interest are recorded and the accepted cases are arranged alphabetically and chronologically in the bibliography.

CASE REPORT

M. S., a sixty-eight year old, single, Russian laborer was admitted to Bellevue Hospital, with a chief complaint of pain in the epigastrium. The family and past histories were irrelevant.

For two years the patient complained of dull epigastric pain which radiated downward over the entire abdomen, with indefinite relationship to meals. Vomiting after meals, diarrhea and bloody stools, anorexia, a slightly productive cough and dyspnea had all persisted for one month. Precordial pain had been present for three days. He complained of nocturia five to six times for a month, and had lost 60 pounds in weight.

Physical examination revealed an elderly, markedly emaciated, dehydrated and dyspneic man. The abdomen was slightly distended. The liver and spleen were palpable but not enlarged. The kidneys were not palpated. There were enlarged inguinal lymph nodes. His blood pressure was 98 systolic and 84 diastolic.

Laboratory Reports. Urinanalysis showed specific gravity 1.010; alkaline reaction; trace of albumin; a few epithelial cells but no pus or red blood cells were reported. Blood count: leucocytes 8200; polymorphonuclears 78, lymphocytes 20, and transitional cells 2 per cent; erythrocytes 5,490,000; hemoglobin 70 per cent.

A provisional diagnosis of carcinoma of the stomach was made. The patient died the following day before further studies could be accomplished.

Autopsy. An emaciated white male measuring 5 feet, 10 inches and weighing about 150 pounds. No abnormal changes referrable to nose, mouth, eyes or ears were found, nor edema, jaundice or enlargement of the superficial lymph glands. The distribution of the hair was normal. The external genitalia appeared normal but the genitourinary organs were of most interest and are described in detail.

The right kidney weighed 140 grams, appeared to be of normal size and was of reddish color. The capsule stripped easily leaving a smooth surface. There were no hemorrhages. On section, the normal markings of the kidney were easily distinguished. The right ureter appeared normal. The left kidney was increased in size and appeared lobulated. The kidney was bluish-red and on section consisted of numerous well separated cystic areas. The parenchyma of the kidney was reduced to a shell and a greenish-yellow exudate was present in the pelvis of the kidney. The ureter and the pelvis were markedly dilated, the ureter being dilated in its entirety. About 8 cm. from its entrance into the bladder there was a thickening of the ureteral wall

consisting of firm, white tissue, with encroachment upon the lumen by this growth. The bladder was of normal size with a slightly

Fig. 1. Low power of diffusely growing epithelioma of the ureter, grade II.

thickened wall. Close to the bladder neck the mucous membrane appeared hypertrophied and there was a region of bluish-black discoloration covered by an area of thick, yellowish exudate. The prostate was increased in size and cut with increased resistance, but appeared normal on section. There was no evidence of local extension or metastases in the other abdominal or thoracic organs.

Summary of Anatomical Diagnosis. Carcinoma of the lower third of the left ureter with dilatation; hydronephrosis of the left kidney; cystitis; prostatic hypertrophy; bronchopneumonia; arteriosclerosis of the aorta; fatty degeneration of the liver, and general emaciation.

Microscopic examination of the primary ureteral tumor showed a background of fibromuscular tissue scattered through which were innumerable islands of tumor cells. The latter, for the greater part, showed large rounded or oval, rather richly chromatic nuclei embedded in a scanty smooth pinkish staining cytoplasm. Among them were a few very large flattened cells with richly chromatic, sometimes lobulated nuclei resting in relatively very large amounts of smooth, bright pinkish staining cytoplasm. The whole represented the remains of cells of the squamous type. The tumor is a diffusely growing epithelioma.

Incidence. The observation here reported is the only instance of proved primary ureteral cancer in the 22,810 autopsy files of Bellevue Hospital from 1904–1935. The clinical files of the urological service of Bellevue Hospital show no records of this rare disease; Pack and Le Fevre in 1930 disclosed no cases observed at the Memorial Hospital of New York, in their study of 16,565 malignant tumors.

Age Period. There were no children or young adults in this series. Zironi's and Scott's reports are the only records of the fourth decade, both patients being thirty-six years of age. The oldest case reported by Richter, was an eighty year old woman. There were 11 cases in the fifth, 10 each in the sixth and the seventh and 6 cases in the eighth decades. The mean age for the entire series was fifty-eight years while the average age was fifty-seven years.

Sex and Color. There were 24 males and 17 females in this series. Rousselot and Lamon reported the only tumor present in a Negro.

Previous Duration of Symptoms Related to Ureteral Cancer. In 7 cases this data is either missing or indefinite; in the remaining 34 cases the average duration prior to diagnosis or operation was fifteen and one-half months. If the cases of Israel and Loewenstein, where the history recorded symptoms for ten years, and the recent report of Cochems and Grauer with a duration of fifteen years are excluded, then we find a period of seven months as the average duration prior to diagnosis or operation.

Israel and Loewenstein in 1911 reported a right sided ureterocele coincident with cancer of the left ureter; the presence of this ureterocele suggests urinary tract pathology of probably long duration. A recent report of my own with combined bilateral ureterocele, ureteritis cystica, and

primary cancer of the right ureter also gave a seven year history of urinary symptoms. Cochems and Grauer's patient, a fifty-one year old woman, complained of aching pain across the right abdomen and the right lumbar region for fifteen years. No hematuria was noted but the right kidney was palpable. The primary cancer, located in the middle third of the right ureter was found associated with leucoplakia and a small calculus. Here again the long duration of symptoms suggests the presence of previous benign ureteral obstruction with subsequent hydronephrosis.

Symptomatology. Pain was the principal symptom which brought the patient for examination, as recorded in 33 cases. In 6 cases the pain was predominantly in the back, and in the cases of Spiess, Schmitt and Glas the provisional diagnosis of "sciatica" was made. Hematuria was noted 21 times. Volante's patient had noted gross bleeding eleven years previously. In 28 instances a palpable tumor mass was noted on examination, which usually proved to be a hydronephrotic kidney above the ureteral obstruction. The primary ureteral tumors per se were not bulky in cross section, although the entire ureter may be involved.

Cystoscopy and Roentgenographic Examination. Cystoscopic examination was performed 22 times and in 16 cases aided directly in establishing the correct diagnosis. The roentgenogram assisted in the diagnosis of associated calculi in 5 of 6 cases. Davy in 1884 found the calculus at autopsy; however, he performed cystoscopy but was unable to obtain urine from the left ureter which later proved to be the seat of primary cancer. Sommer in 1932 was confused by the presence of a calcified ovary which existed on the same side as the primary tumor. Severe hemorrhage followed manipulation in Scott's and Hunter's cases which directly aided in the correct diagnosis.

Diagnosis. In 9 reports a correct clinical diagnosis was made before operation or autopsy. The diagnosis of malignancy was made in the following cases: Ureteral tumor, eight times; osteosarcoma of the pelvis, twice; kidney tumor, twice; bladder tumor, twice; kidney pelvis tumor, once; and cancer of the stomach, once.

The 2 cases of Hektoen and Butler suspected of being primary sarcoma of the pelvis later proved to be tumors of the lower third of the ureter, both on the right side, with secondary extension to the bony structures. Our own case was in extremis and the provisional diagnosis of cancer of the stomach was made from the history of predominantly gastrointestinal symptoms. This symptom complex is commonly confusing in the presence of obstruction to the upper urinary tract.

Operative Procedure. Surgical removal was not attempted in 20 instances, due either to inability to make a correct diagnosis or the extremely poor condition of the patient on admission. Nephrectomy only was done by Gerstein. Nephrectomy and ureterectomy was done five times, while nephrectomy and splenectomy was done by Israel. Partial low ureterectomy only was performed in Snyder and Wood's case because the condition of the patient prohibited further procedures. Hunter performed partial ureterectomy without nephrectomy and successfully removed the entire tumor. Subsequent death was due to a colloid carcinoma of the stomach six years later. Exploratory kidney operations were done six times. Exploratory laparotomy, cystotomy, bladder cauterization and prostatectomy were performed once each. In 2 cases, bladder tumors were considered as extensions of the primary ureteral cancer. In the light of newer interpretation these tumors should be considered as the extensive or diffuse simultaneous appearance of the same process and not a metastasis or implantation.

Pathology. Side Involved. The seat of the primary tumor was located 17 times on the right and 24 times on the left side The right ureter was involved twice in the upper third, 6 times in the middle, and 16 times in the lower third. The left ureter

showed lesions twice in the upper third, twice in the middle and 13 times in the lower third. The middle and lower thirds were involved in one extensive tumor twice on the right and once on the left side. Ureteral anomalies were not common. Jona reported a papillary tumor present in a diverticulum of the lower third of the left ureter about 1.5 cm. in diameter.

Local Extension and Metastases. In the 2 cases of Jona and in the case of Ascoli the tumor was limited entirely to the ureter without local extension or metastases. In 13 instances there were no local extensions while in 20 cases there were localized periureteral infiltrations. The bladder was involved five times while the seminal vesicle and the dorsal vertebras were each involved once.

Metastases. There were no metastases present in 11 autopsies. The retroperitoneal lymph glands were involved 12 times; the liver 9 times; the lungs 8 times; the kidneys 5 times; while spleen and bones were affected 3 times each, (ilium once and vertebras twice); peritoneum, appendix, pararectal lymph glands, Fallopian tubes, vagina, pancreas, adrenals, skin, the vena cava, and the ureter of the opposite side once each. It will be noted that in only 8 cases the metastases extended above the diaphragm.

Microscopic Diagnosis. Various terms were used to describe the non-papillary flat tumors which occurred in 7 instances. The distinctly squamous tumors numbered 15, while carcinoma and adenocarcinoma were recorded twice each. Carcinosarcoma was diagnosed once in Renner's case. Fourteen tumors were described as papillary in structure.

The tumors coincident with calculi are of special interest as their presence as a source of chronic irritation is beyond doubt. The association of calculi and tumors was noted in 15.3 per cent of the series. Six such reports are given by Davy 1884, Zironi 1909, Paschkis 1910, Volante 1927, Rousselot and Lamon 1930, and Cochems and Grauer, 1935. In the reports of Davy and Paschkis the tumors were papillary while the remaining 4 were flat or squamous in structure.

MacMillan and I found the closely related tumors of the renal pelvis to be highly malignant and associated with calculi in 52 per cent of 57 cases. We feel that the early removal of impacted calculi should be considered as a prophylactic procedure.

Associated Diseases and Double Primary Tumors. Cross found pulmonary tuberculosis present in the autopsy of his forty-eight year old male patient. Microscopic evidence of syphilis was observed in Renner's seventy-one year old male patient. Ockerblad and Hellwig stated their patient had been cured of an epithelioma of the nose five years previous to operation, although microscopic evidence was not definitely stated. In both Renner's, and Cochems and Grauer's reports associated spindle cell sarcoma of the bladder was noted, while Rousselot and Lamon observed a dermoid cyst of the ovary and adenomyosis of the Fallopian tubes in a forty-eight year old Negress. Hunter's patient, a male, forty-three years of age, lived six years after operation for a papillary carcinoma of the right ureter, only to die of a perforated colloid carcinoma of the stomach. Scott's second case died following an exploratory abdominal operation with colostomy about three and a half months after nephrectomy for hydronephrosis. Autopsy disclosed two primary cancers; one of the transverse colon and the other of the lower third of the right ureter.

These cases of multiple primary tumors clearly show the necessity for complete autopsy studies. Six cases or 15 per cent is unusually high in any series of autopsy statistics.

Prognosis. There are 6 records with incomplete data in regard to the duration of previous symptoms. Of the 35 cases with complete data, 19 or 54 per cent lived a total of two to nine months. Excluding the 2 cases with long histories there were

14 patients, or 40 per cent who lived from one to three years. The total length of life of the entire series from the onset of the first symptom to the time of death was twenty-one months. With the exclusion of the reports by Israel and Loewenstein (ten years) and Cochems and Grauer's (fifteen years) the total length of life was exactly one year.

The surgical figures of Scott also showed a postoperative mortality of 27 per cent, and the follow-up revealed that more than 50 per cent died within twelve months.

Scott's questionnaire on surgical end-results revealed the fact that 2 and probably 3 patients had survived removal for primary ureteral cancer more than five years after operation. Kraft in 1922 reported the result of nephroureterectomy for multiple papillary epithelioma in a fifty-two year old woman. Follow-up in July, 1932, revealed the patient alive eleven years after operation. Crance and Knickerbocker in 1924 made a preoperative diagnosis of primary tumor of the lower right ureter in a forty-three year old woman who had symptoms for one year. Ureteronephrectomy revealed an epithelioma, and the patient was reported well eight years later. Stewart's patient in 1926, a seventy-five year old woman lived four years after nephrectomy and ureterectomy for papillary cancer of the middle right ureter. Death occurred from pneumonia and cardiac failure. While no autopsy was performed, the disease was probably arrested.

The only addition to these successful surgical results is the report of Hunter in 1935. His forty-three year old male patient survived ureterectomy for papillary carcinoma of the lower third of the right ureter for six years and three months from the onset of the initial hematuria. Death was due to a colloid cancer of the stomach with metastases to the omentum.

It should be noted that 3 of these 4 cases were papillary carcinomas and that in all reports the authors suspected the correct diagnosis preoperatively and followed well planned surgical procedures.

SUMMARY

The literature of 40 autopsy cases of primary cancer of the ureter is reviewed to which is added the only report from the Bellevue Hospital files of 22,810 protocols. There were 24 males and 17 females. The peak of the age incidence is in the fifth decade with the average at fifty-seven years. All patients except one were white. The average duration of symptoms previous to observation was fifteen and one-half months. Two reports of extremely long previous histories are given for ten and fifteen years, respectively. Pain, hematuria, and palpable tumor were the three most constant findings in the history and physical examination. Backache was the chief symptom in 6 cases. Cystoscopy and roentgenogram directly aided in making a correct diagnosis 16 times, while associated calculi were found 6 times, or 15 per cent. The tumor was right sided 17 times and on the left 24 times. The lower third of the ureter on both sides was the seat of the tumor in 77 per cent of the series, in 2 cases being localized entirely to the ureter. In 13 reports there was no local extensions but distant metastases were present, while 11 showed no metastases. The metastases were variously distributed to the abdominal viscera and bone, but only 8 times extended above the diaphragm. Twenty-seven primary tumors were flat or non-papillary while 14 were papillary. Six or 15 per cent showed double primary tumors. Nineteen or 54 per cent of patients lived from two to nine months from the onset of the first symptom, while 14 or 40 per cent lived from one to three years. One six year survival is added to 3 previously reported surgical results where apparent successful removal was accomplished.

I wish to thank Dr. Douglas Symmers for his review of the autopsy material.

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HYDRONEPHROSIS DUE TO BALL-VALVE OBSTRUCTION FROM BULLET LYING FREE IN RENAL PELVIS

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RECENTLY Campbell[1] reported a case of a hydronephrotic kidney caused by a papilloma of the renal pelvis. Occasionally a stone lying free in the renal pelvis may, through a ball-valve action, cause obstruction to the outflow of urine, producing a hydronephrosis, but in the case here presented this was caused by a bullet.

A female, aged fifty-nine years, was admitted to the Lucas County Hospital on January 24, 1935, complaining of pain in the abdomen and back. Seventeen years ago she was shot in the right upper portion of the abdomen; an operation was performed immediately but the bullet was not found. She made an uneventful convalescence. A few years later, however, she began to have frequency, burning on urination, and pain in the lower abdomen, especially on urinating. There was no hematuria at this time but she thinks that there was blood in her urine directly after the injury.

Eleven days ago she began to have a sharp, severe pain in the right lumbar region with radiation toward the bladder and an exacerbation of the dysuria.

Inspection revealed a fairly well nourished female apparently suffering pain. Examination revealed a few rales in the right lung, tenderness in the region of the right kidney, and a scar over the upper right rectus. The systolic blood pressure was 140 mm. of mercury and the diastolic 80 mm. The urine revealed a trace of albumin and many pus cells. The hemoglobin was 91 per cent, red blood cells 4,400,000, white blood cells 6200. The Kline test was negative. The non-protein nitrogen was 30 mg. per 100 c.c. of blood. The phenolsulfonphthalein test showed 75 per cent elimination in two hours.

A roentgenogram of the kidneys, ureter, and bladder revealed a shadow in the region of the right kidney, apparently a bullet, and another shadow in the right lumbar region below the crest of the ileum. Cystoscopic examination showed a diffuse cystitis. Both of the ureteral openings were edematous. A No. 6 catheter was passed easily up the left ureter and a left pyelogram was made which was normal. An intravenous urogram demonstrated a normal left kidney with no appearance on the right side. Another cystoscopy was then done and a Braasch bulb placed in the lower right ureter and a right urogram made. This showed the lower shadow to be included in the lower ureter and the upper shadow to be included in the upper ureter and renal pelvis. A diagnosis was therefore made of a calculus in the lower right ureter and a bullet in the region of the right kidney.

Operation was performed on August 15, 1935 employing spinal anaesthesia. An incision was made in the right costovertebral angle downward and medially to a point about one inch above the anterior-superior iliac spine and the kidney was dissected free from its fatty capsule. In attempting to separate the fat from the region of the pelvis of the kidney considerable scar tissue was encountered, probably due to the former gunshot wound. By means of blunt and sharp dissection the kidney was isolated except for its pelvis and inferior and superior poles. The tissue holding the poles was doubly clamped and divided, the distal ends being tied with No. 2 plain catgut. By means of blunt dissection the course of the ureter was uncovered well down toward the pelvic brim. About the level of the pelvic brim a stone was palpated, which was about 1½ inches long and ½ inch in diameter. An incision was made over this stone in a longitudinal direction, the stone removed and the incision closed. About 3 inches above this opening the ureter was isolated, triply clamped and divided. The distal portion was doubly tied with No. 2 chromic catgut. The ureter was then freed in its proximal portion until the pedicle of the kidney was exposed. An accessory vessel was found

[1] CAMPBELL, MEREDITH F. Hydronephrosis due to ball-valve obstruction by papilloma at ureteropelvic junction. *Am. Jour. Surg.*, August, 1936. Page 291.

near the inferior pole of the kidney; it was doubly clamped and divided, the lower clamp being left in place. A large kidney clamp was then placed on the pedicle and two additional clamps were placed distally, clamping off the width of the pedicle. The kidney was then excised distal to the clamps. A rubber dam drain was placed well down in the right flank. Using No. 2 chromic catgut an attempt was made to close the transversalis, locking the sutures around the drains and clamps. The tissue was, however, so friable that this line of suture was practically worthless. The internal oblique muscle and fascia were closed with No. 2 chromic catgut; the external oblique and latissimus dorsi muscles were likewise approximated with No. 2 chromic, and three silkworm gut tension sutures were placed. Clamps were each tied together with No. 2 plain catgut to prevent their opening. The skin was closed with clips; the dressing was secured by adhesive tape. A pasteboard box covering for the four clamps was secured by tape. The clamps were removed in seventy-two hours.

The pathologist's report is as follows: The specimen consists of a kidney with considerable surrounding fat, and measures 8 by 3.5 by 2.7 cm. The renal pelvis bulges downward and is nearly as large as the rest of the kidney and a hard object, resembling a bullet, moves freely around in the pelvis. There is a definite hard scar on the greater curvature, thought to be the point at which the bullet entered the kidney. On incising the kidney in the usual manner and opening the pelvis, the bullet is readily released, since it lies free in the pelvis. It appears to be a .38 caliber bullet, the diameter is about $\frac{5}{16}$ of an inch, the length is about $\frac{5}{8}$ of an inch, and the bullet is only slightly distorted. The scar on the cortex which is suggestive as being the point of entry of the bullet contains a subcortical stone, the size of a grain of puffed wheat, and there is no track to indicate the course of the bullet. There is another stone the same size in a nearby calyx. The kidney substance shows areas of chronic infection with very little good renal tissue remaining. The pelvis shows several inflammatory patches.

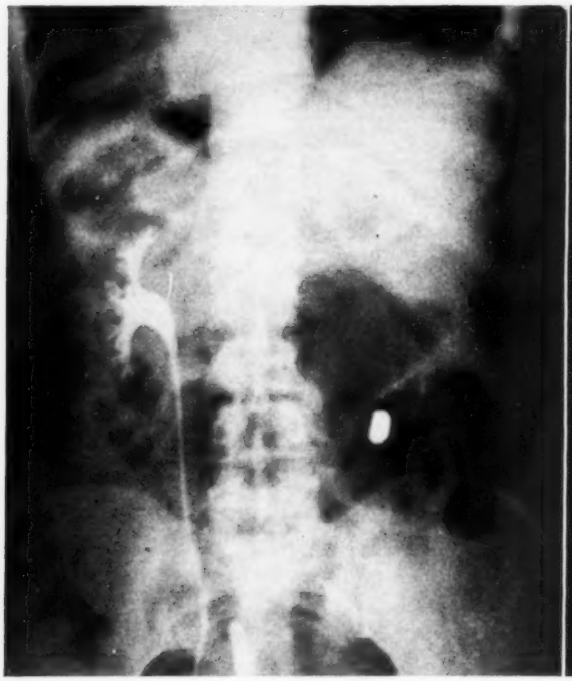

Fig. 1. Normal urogram of left kidney with bullet appearing in the upper right abdomen.

Fig. 2. Retrograde urogram of right kidney and ureter showing inclusion of bullet and suspicious shadow (stone) in lower ureter.

There was received separately a stone somewhat the shape of the bullet, measuring 2.5 cm. in length and 1.3 cm. in diameter and said to have been in the lower ureter.

Diagnosis: Hydronephrotic kidney with .38 caliber bullet lying free in the pelvis; ureteral stone.

The convalescence was uneventful and the patient left the hospital in good condition and relieved of her former symptoms.

A bullet lying free in the pelvis of a kidney is, to say the least, unusual. Whether the bullet entered the kidney at the time of the shooting or burrowed in later is problematical. I believe, however, in view of the hematuria, which probably occurred, that the kidney was injured in the first instance. The occurrence of the stone lower down in the ureter below the obstruction may have some relationship to the stasis, infection and chemical changes in the urine that undoubtedly resulted from the bullet in the pelvis.

Morris[2] relates several cases from the Napoleonic wars in which there was a bullet wound of the kidney and weeks or months later the patient passed shreds of clothing from the urethra. The records of the World War in regard to bullet wounds of the kidney have not been reviewed but it would be interesting to learn if this type of injury has occurred.

SUMMARY

The article is a report of a woman who years before was shot in the abdomen. The bullet evidently lodged in the renal pelvis and through a ball-valve action caused hydronephrosis and destruction of the kidney.

[2] Morris, Henry. Diseases of the Kidney and Ureter. Vol. 1, pp. 230 and 231.

PLASTIC RESTORATION FOR LOSS OF ALL FINGERS OF BOTH HANDS

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THE loss of all the fingers of both hands is a disabling occupational injury. All the delicate and dextrous movements which depend upon the highly specialized mechanism of normal finger motion are permanently lost. The stump of either hand alone is useless and to perform any kind of work it is necessary to place the two together. With the two stumps in apposition many gross acts may be accomplished but those requiring finer movements are impossible.

If we wish to obtain additional function it is possible by plastic surgery to transform the metacarpals into fingers. Several factors that have an influence in the results must be taken into consideration when planning the operations. In the first place the mobility of the newly formed member will naturally be restricted on account of the limited range of motion that exists at the metacarpocarpal joints. Then there is usually a considerable amount of scar tissue present that adds to the difficulty of the operation and hinders motion. It is also important that there be sufficient good functionating muscle to control the movement of the new digit and there should be a normal sensory nerve supply.

The brief history of a boy with loss of all the fingers, together with an outline of the operation will be reported.

E. C. L., aged eleven years, male, at the age of one and one-quarter years, or nine and three-quarter years ago, fell into hot ashes and received severe burns of both hands. Nothing is known about the immediate or subsequent treatment. The fingers were burned at their ends and became buried beneath a covering of skin. During the ten years following the accident the patient became quite adept in the use of the two stumps of hands when held together and could cut his food, comb his hair and do many useful things in a remarkable manner. With one stump alone he could do very little.

Examination. There are no visible fingers on either hand. The burnt off stumps of the fingers are buried beneath a covering of skin and fibrous tissue (Fig. 1). On the left hand there is good extension and flexion of the wrist. When the patient attempts to move the stumps one can feel beneath the skin the remnants of the first phalanges of the third, fourth and fifth fingers moving fairly well at the metacarpal-phalangeal joints. There is little motion between the first phalanx on the second metacarpal bone but practically none in the first metacarpal-phalangeal stump. On the right the wrist is held in a position of about 35 degrees of flexion and ulnar deviation. The buried remnants of the fingers are much more fixed than on the opposite side and several of the stumps appear to be fused to each other. In the roentgenogram (Fig. 2) one sees several of the first phalanges present. They are flexed at the metacarpal-phalangeal joint.

Treatment. There was a total of thirteen anesthetics and several operations were carried out at the same time.

First operation, December 14, 1932, right hand. (1) Release of wrist contracture. The scar tissue was resected and the gap filled with a split skin graft. (2) Plastic to form thumb.

First Stage. Dorsal and plantar flaps were outlined between the first and second metacarpal bones and a cleft was formed by cutting down between them. The flaps were sutured in place and the remainder of the gap was filled in with split skin grafts.

Second operation, January 30, 1933, right hand. Plastic reconstruction of the thumb: Flaps corresponding somewhat to a Didot operation were formed between the first and second metacarpal bones. The defects were filled in by split skin grafts.

Third operation, March 8, 1933, left hand. Plastic between the second and third metacarpal bones: An incision was made between the second and third metacarpals. The defects

in the skin were covered by full thickness grafts obtained from the abdomen.

Fourth operation, April 7, 1933, right hand.

FIG. 1. The appearance of the hands before operation; contracture of right wrist and complete absence of fingers on both hands.

Deepening the cleft between the first and second metacarpal bones: The cleft was deepened and the defects in the skin were covered by full thickness grafts removed from the right thigh.

Fifth operation, July 5, 1933, left hand. Deepening of the cleft between the first and second, and between the second and third metacarpal bones: The clefts were deepened by cutting at the base of the already formed clefts. The defects were covered by shifting flaps of skin, full thickness grafts and split skin grafts.

Sixth operation, August 11, 1933, right hand. Deepening of a cleft between the thumb and the adjoining stump: Flaps were outlined and after cutting down between the metacarpal bones the flaps were shifted to fill in as much as possible of the gap. The remainder of the defect was filled in with split skin grafts.

FIG. 3A. Four rudimentary fingers were formed on the left and one on the right hand. There was good mobility between the digits.

Seventh operation, September 25, 1933, right hand. The cleft was again deepened between the thumb and the adjoining stump and the defect was filled in by shifting flaps and full thickness grafts. A tubular graft was prepared on the abdomen at the same time.

Eighth operation, November 10, 1933, left hand. (1) Formation of cleft between the third

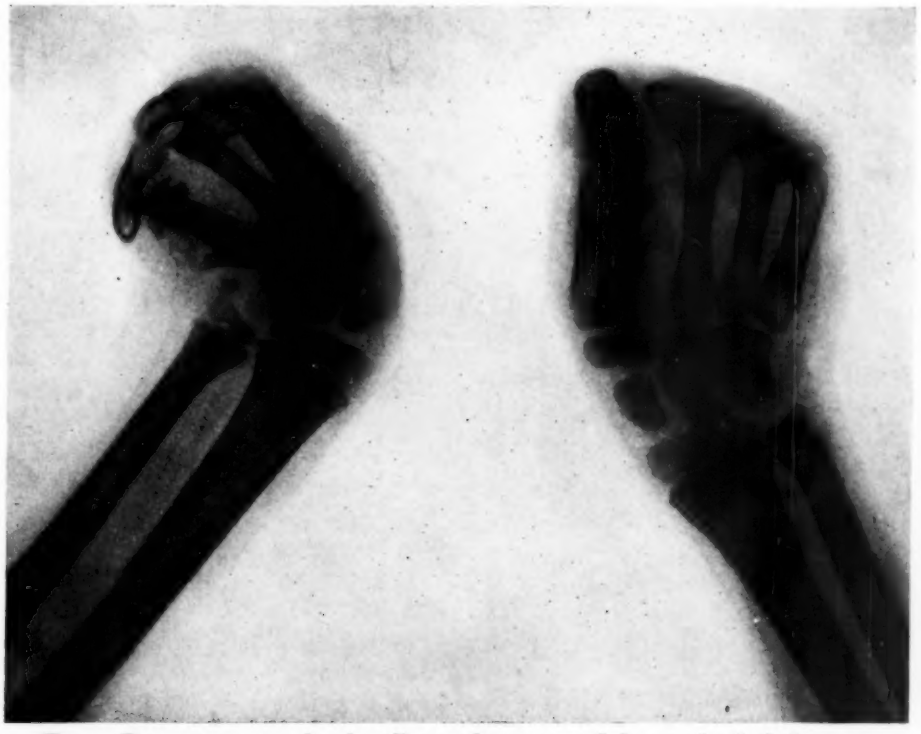

FIG. 2. Roentgenogram showing distorted remnant of the proximal phalanges.

and fourth metacarpal bones and (2) transference of one end of the tubular graft: The lower end of the tubular graft was freed and transposed to a bed prepared on the left hand. The bed for its reception was formed by cutting between the third and fourth metacarpal bones.

Ninth operation, December 13, 1933, left hand. Transference of the remaining end of the tubular graft: The remaining attachment of the tubular graft was excised and sutured.

Tenth operation, March 12, 1934, left hand. Transference of part of the tubular graft: The tubular graft was cut into two pieces. The part toward the palm was opened up. The scar tissue was resected from the palmar surface of the hand and the opened up tubular grafts were sutured into the defect.

FIG. 3B. Showing reconstructed palmar surface of the left hand and flexion of thumb of the right hand.

FIG. 4. Using knife and fork.

Eleventh operation, April 8, 1934, left hand. Deepening of the cleft between the second and third and the third and fourth finger stumps: Portions of the tubular graft were transferred to the gaps that were formed by cutting deeper between the second and third and third and fourth metacarpal bones.

Twelfth operation, May 16, 1934, left hand. Plastic of the palm between the new stumps: Portions of the tubular grafts were released at advantageous points and shifted between the various stumps and into the palm to replace contracted tissue.

Thirteenth operation, June 27, 1934, left hand. Plastic on the stump; full thickness graft to the palm: Further shifting of flaps was done about the bases of the stump. A defect that was left after this shifting of flaps was filled in by a

full thickness graft that was removed from the left thigh.

The final result was the forming of a rudimentary thumb on the right side, giving the patient a boxing glove or mitten type of hand (Fig. 3). He attained good movements of the newly formed thumb and could do many useful things such as writing, combing his hair and feeding himself. It was thought inadvisable to attempt to make any other digits on this hand because of the fixation of the joints and the excessive scar tissue.

On the left hand a rudimentary thumb and three fingers were constructed (Fig. 3). Although they appeared thick and bulbous he had very good control over them. He could write, comb his hair, assist in cutting his food and employ his fingers for many useful purposes (Fig. 4). He was able to hold a golf ball in his left hand (Fig. 5). He was learning to lace his shoes and could almost tie a bow knot when he left the hospital.

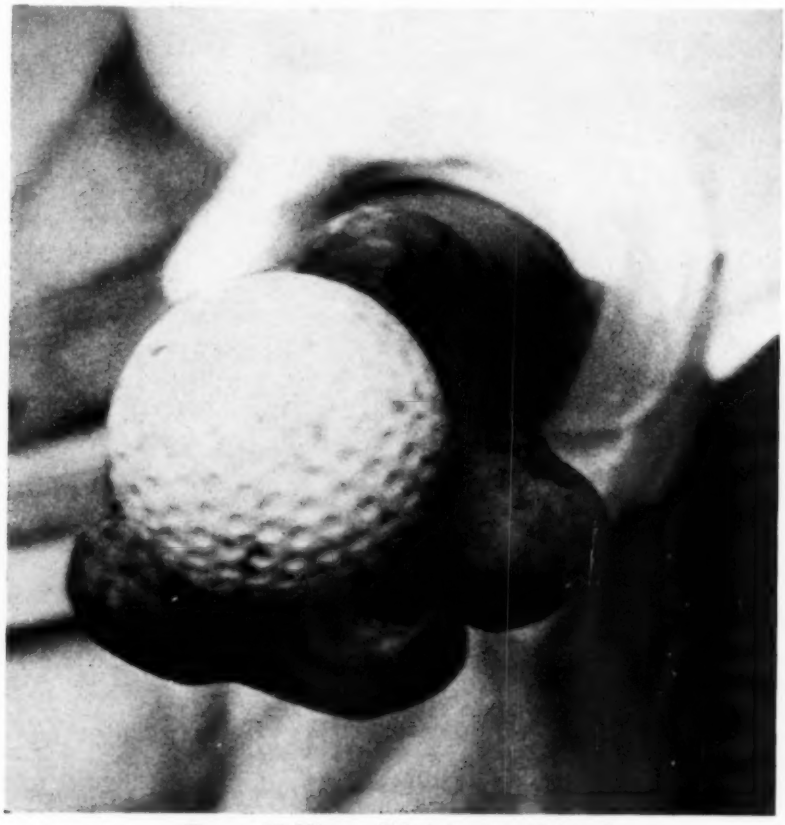

Fig. 5. Holding golf ball in the left hand.

COMMENT

There should be an increased length of the digits as the metacarpals grow and a decrease in the thickness of the members. There is often a tendency for the web to creep up as after syndactylism operation but this did not take place during the first year. With use the range and freedom of motion should increase.

One of the main difficulties encountered was the tendency toward contracture in the palm following each operation. With each addition of a digit there was an increasing contracture tending to pull the fingers toward the middle of the palm and restrict motion. Fortunately each finger maintained its independent motion after the numerous operations. There were no gross sensory disturbances or tendency toward trophic changes.

SUMMARY

The loss of all the distal portion of the fingers on both hands with the forming of a pannus of skin over the stumps furnished the interesting clinical condition reported in this paper. The partial restoration of four digits on one hand and one on the other, allowing independent use of each hand with an increase of function, was obtained by plastic operations.

BLANK CARTRIDGE CANNON SHOT WOUND OF LEG*

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OCCASIONALLY in civil practice surgeons have an opportunity to treat wounds such as occur in military service. It may be of interest, therefore, to report a case in which a thirteen year old boy had a serious cannon shot wound of his left leg.

FIG. 1. Wound of lower leg with exposed shaft of tibia denuded of periosteum.

In July, 1933 while at camp in Maine, W. C. O. fired a small, marine cannon as the morning salute to the raising of the flag. The cannon was one of those small type that one sees on board ships and which are used for saluting shots. They are loaded with shot composed of powder and cotton wadding. How this accident occurred is not exactly known but the fact remains that this chap in some manner pulled the cord on the trigger at the moment that his left leg, at the junction of the middle and lower thirds of the mesial aspect, was directly in front of the muzzle of the cannon. The skin, portions of the muscles and all of the covering over the tibia including the periosteum, were blown away by the close range explosive force of the shot. He was rushed to a nearby hospital in Lewiston, Maine, where first aid was given.

Dr. Walter H. Galland of New York was called and upon his arrival in Maine he realized that an extensive reconstruction operation would be necessary to repair the defect of the leg and it was through his courtesy that I operated upon this patient with him. Tetanus antitoxin was given immediately and the patient was transferred to New York where he arrived forty-eight hours after the injury. The leg presented an area of the tibia about 3 inches long completely denuded of its periosteum and the bone charred and burned by the powder (Fig. 1). The surrounding area for about 2 inches beyond the wound margin showed the typical effect of a close explosive injury. The tissues were black, necrotic and devitalized and burned by the powder. The muscles on the inner aspect were exposed and likewise black and charred.

An immediate débridement operation was performed under avertin anesthesia. The leg was thoroughly scrubbed with ½ per cent lysol and green soap getting rid of as much powder markings as possible. Thereupon, excision of all of the devitalized tissue was done, removing skin, aponeurosis and superficial muscle and cutting through normal healthy periosteum to get a clean edge. This resulted in a wound approximately 6 inches long and 4 inches wide and left a large, exposed area of tibia denuded over half its extent of periosteum. The wound was immediately treated with Carrel-Dakin solution.

The next days were anxiously watched to make sure that no infection along the fascial planes of the leg would occur. Another dose of tetanus antitoxin and gas bacillus serum were given. Fortunately, no infection occurred. Smears of the wound were made and watched for evidence of bacteria.

Dr. Galland and I believed that the sooner the denuded and charred tibia would be covered with normal tissue the greater would be the

* Presented before the Surgical Section of the New York Academy of Medicine, May 4, 1934.

chance of maintaining or restoring its vitality. The medical and surgical consultants agreed with this and on the third day after the débridement and even before the wound smears were free of bacteria a reconstruction operation was undertaken, under avertin anesthesia.

Fig. 2. Left leg held in place by plaster-of-Paris cast with window cut out to expose pedicle and flap.

Fig. 3. Close up view of flap healing in place. Thiersch graft healed on undersurface of pedicle. Granulating wound at lower end of flap where sutures were removed at primary operation to release the tension.

The operation was performed on the Albee fracture table. Of the greatest assistance and a point which we consider of the most vital importance, was the fact that Dr. Galland was associated with us at this operation. The success of these types of cases depends as much on keeping the legs properly immobilized as it does on the operative procedure. It was, of course, essential that this large defect be covered with a pedicle flap composed of normal skin and subcutaneous fat. A Thiersch skin graft or a full thickness graft would never take over this area of denuded and partially devitalized bone. To take a pedicle flap from the posterior aspect of the same leg was impossible as the width of the flap necessary could not be obtained from the posterior aspect of the same leg. Therefore, a pedicle tongue flap from the opposite thigh had to be used. A model of the defect was cut beforehand and was studied carefully so that we knew exactly where to place the pedicle of the flap on the right thigh. A number of important principles had to be kept in mind in planning the operation.

1. The flap had to be somewhat larger than the actual defect.

2. The pedicle had to be long enough to allow the flap to jump across from the thigh to the leg and lie in place without tension.

3. All raw surfaces should be immediately covered with Thiersch skin grafts to obtain complete primary union and to have no resulting granulating wound. This was especially important during the heat of the summer when maggot infection of a secreting wound in a cast occurs so easily.

4. The two legs should be held rigidly together by plaster cast to prevent any motion of the flap or pull on the pedicle.

The large flap from the anterior and outer aspect of the lower portion of the right thigh was then raised with the base of the pedicle above. Skin, fat and superficial fascia were included in the flap exposing the fascia lata over the muscles of the right thigh. After the flap and pedicle were raised the left leg was crossed to test the flap and see whether it would fit into the defect and also whether the pedicle was

long enough. After this was ascertained the skin margins on the right thigh were slightly undermined to permit any decrease in the size

Fig. 4. Right thigh from which pedicle flap was taken. Thiersch graft on outer and inner aspect of thigh. Picture was taken one month after operation.

of the defect by suturing the skin edge to the fascia lata. It was impossible to decrease this wound very much in size. The entire raw surface was then covered with Thiersch skin grafts carefully taken from the left thigh. The edge of the wound was covered with a narrow strip of tampon on which some 1 per cent fuchsin ointment had been applied. Where Thiersch grafts overlapped one another a similar strip of gauze was laid. The whole grafted area was then covered with gauze which was most meticulously strapped with sterile adhesive to prevent any shoving of the Thiersch skin grafts. This was necessary as we knew that the grafted area could not be dressed for some time. In another similar case it would be well to place a rubber sponge between the leg and the thigh to act as a soft cushion or a pedestal on which the leg rests.

A Thiersch graft was also placed on the raw undersurface of the pedicle of the flap and held in place with sutures of interrupted plain catgut. The left leg was then brought across the right thigh and the flap found to fit perfectly into the defect. It was sutured in place with interrupted black silk sutures except where the pedicle crossed the defect. After suturing the flap in place it was found that its lowermost portion became totally blanched and would be in danger of necrosis. Several sutures, therefore, on the lower portion of the flap were removed, releasing the pressure and allowing some of the fat to be exposed and to be allowed to heal by granulation. Later observation showed that this was most important as we feel quite certain that at least one-quarter of the flap would have been lost if these sutures had not been removed. As soon as sterile dressings had been put in place Dr. Galland applied a plaster spica around the pelvis, the flexed left knee and the right leg down to and including the right foot (Fig. 2).

We wish to emphasize once more that in these types of cases the closest coöperation between a surgeon and an orthopedic surgeon is of utmost importance for their successful conduct. The many buttresses and reinforcements, windows, etc., which Dr. Galland put into this cast made it possible to have the entire flap with its pedicle exposed so that close observation and dressings were technically possible (Fig. 3). Naturally a cast of this type was quite heavy and the patient was placed on a fracture bed in which the mattress could be lowered, permitting bedpans, etc., to be raised under the patient. It also permitted the turning of the patient from side to side, allowing the projecting knee and foot to pass between the canvas straps of the fracture bed. It was necessary to improvise a large rubber tube which had to be passed between the cast and into which the patient urinated, as it was impossible to bring a urinal up to the penis. The following three weeks were difficult for the patient as well as for the nursing staff and the doctors. To lie immobilized in this position during the heat of the summer was no trifle for this young man and he deserves a great deal of credit and commendation for his coöperation.

The wound healed by primary union including the graft on the pedicle of the flap. On the twentieth postoperative day the cast was removed under avertin anesthesia and the pedicle divided, the flap implanted and sutured to the wound margin of the left leg. The only abnormal condition found upon removal of the cast was a small pressure ulcer on the outer side of the left leg where the leg had been in contact

with the padded dressings over the Thiersch grafted area of the right thigh. It was decided at this time to do a further plastic operation on the right thigh after excising the Thiersch graft from the remainder of the pedicle and from the thigh so that the remaining pedicle could be replaced into its former position. In order to avoid a non-elastic Thiersch grafted area over the quadriceps extensor muscle directly in front of the right thigh and to allow for better function and elasticity on flexion of the right knee, a further pedicle tongue flap from the inner aspect of the right thigh with the base of the pedicle above was raised and swung across the front of the right thigh. The resulting defect on the inner aspect of the right thigh was then decreased in size by undermining the skin edges and suturing them down to the fascia. The remaining defect was covered with a Thiersch skin graft from the left thigh. This re-arrangement of the tissues of the right thigh then resulted in non-elastic Thiersch grafts on the inner and outer aspects of the right thigh with practically normal elastic skin over almost the entire extent of the front of the right thigh (Fig. 4). All of these wounds healed kindly. The main flap over the gunshot wound area remained totally viable and healed normally in place (Fig. 5). The tibia was carefully watched for the possibility of sequestration of the cortex since the cortex had been burned by the heat of the explosion and denuded of periosteum. Repeated observation by *x*-ray film from month to month showed that the early covering of the bone with the pedicle flap made it possible for the bone to recover and no sequestration took place (Fig. 6).

A B

Fig. 5. A, anterior view and B, lateral view of flap healed in place one month after operation.

In the immediate convalescence following the last operation there was some difficulty in regaining flexion and extension of both knee joints and of the ankle joints but this rapidly improved with massage and physiotherapy.

It is now three years since the injury and the operation; the patient has been totally restored to normal function and is able to partake of every form of athletic exercise and goes about his work and play the same as he formerly had done.

CONCLUSIONS

This case is reported to bring out a number of points in the treatment of such injuries requiring reconstruction operations.

1. Early and thorough débridement of all injured and destroyed tissues;
2. Early covering of exposed bone especially if denuded of its periosteum;
3. Careful planning and measurement for the proper preparation of the flap and its pedicle;
4. The immediate complete covering of all raw surfaces with Thiersch skin grafts;
5. The vital importance of the association of the surgeon and an orthopedic surgeon in order to get the best complete immobilization of the extremities and still make it possible to expose the flap and dress the wound by properly placed windows in the cast, etc.
6. The covering of the anterior portion of the thigh with normal elastic tissue rather than non-elastic Thiersch graft; and
7. The necessity of close coöperation and teamwork between surgeons, nursing staff and patient.

A B

Fig. 6. A, tibia one month after being covered with flap; B, tibia six months after operation.

INTESTINAL PROLAPSE THROUGH ENTEROSTOMY FOLLOWING RAMMSTEDT OPERATION

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A MALE, four weeks old, breast fed infant was admitted to the St. Elizabeth Hospital, service of Dr. E. H. Flinn, because of projectile vomiting which began twelve days previously. The baby's abdomen was round and firm; after feedings peristaltic waves were visible passing along the fundus and the cardia, and ending abruptly at the pyloric end. The infant's weight was 6¼ pounds (2.81 kg). All non-essential features are omitted for the sake of brevity. The *x*-ray study of the gastrointestinal tract showed a marked retention of the barium suspension in the stomach, such as would be due to a hypertrophic pyloric stenosis. As the the S.M.A. formula given by gastric gavage was not retained and belladonna had no effect, the infant was referred for operation. A typical Rammstedt operation was performed under local anesthesia, care being taken not to injure the mucosa. The patient was given father's blood intramuscularly, in addition to subcutaneous injections of glucose and saline solution. The vomiting, no more projectile in character, occurred a few times in course of the next two days and then ceased completely. As the mother's milk was very scanty, it was supplemented by human milk furnished by the Health Department of the city of Chicago.

Eight days after the operation the abdomen became distended and the complexion of the infant turned sallow. In course of the following day the distention assumed threatening dimensions; the overlying skin was as thin as tissue paper and large subcutaneous veins were visible on the chest and abdomen. Flatus were passed freely, defecation was normal, vomiting was absent, the temperature never rose above 100.5 degrees. In spite of such a confusing picture, a suspicion of a paralytic ileus was considered as justified and after a consultation an ileostomy was performed under local anesthesia nine days after the Rammstedt operation. The after-treatment consisted of repeated blood transfusions, hypodermoclysis, injections of pitressin and prostigmin and instillations of saline solution through the catheter placed into the enterostomy opening. The infant was exposed daily to ultraviolet light and was given drysdol.

The operation apparently did not accomplish a decompression. The abdomen remained extremely distended although flatus and feces were passing freely through the artificial stoma and not through the rectum. The respirations were labored. A flat *x*-ray picture of the abdomen taken eight days after the second operation was reported as follows: "There is a sharply defined pocket of gas in the right upper quadrant, which appears to be free air under the right diaphragm; the pocket displaces the liver downward. The loops of bowel are all collapsed. As a second guess, the findings may be due to a distended, obstructed colon in the hepatic flexure, but the pocket seems too sharply defined." As various conservative measures did not bring any improvement and the infant became highly dehydrated, sixteen days after the enterostomy a needle was inserted under fluoroscopic control under the costal arch in the right axillary region and thick pus aspirated. An incision was immediately made and a rubber drain inserted into the subphrenic abscess cavity. The latter gradually became obliterated and the fistula closed spontaneously after the purulent discharge disappeared.

As the distention of the abdomen subsided only partially and the rectal examination revealed an obstruction 5 cm. above the anus, a diagnosis of a pelvic abscess was made. Four days after the drainage of the subphrenic abscess a mosquito forceps was inserted through the rectum and a cavity opened, from which thick pus escaped. In the course of the following few days the same procedure was repeated several times to maintain drainage. The purulent discharge per rectum stopped after a few days but no feces were passing through the anus. A rectal examination disclosed a stricture, apparently caused by adhesions. Daily gentle dilatations of the stenosis with Hegar's cervical dilators were instituted. Feces began passing through both the rectum

and the artificial stoma in approximately equal amounts but a new complication appeared. The posterior wall of the intestinal loop, attached to the abdominal wall began prolapsing through the enterostomy every time the child cried. Within a short time the evisceration not only became stationary but increased in size. After a few days the sausage-shaped protrusion in the right lower quadrant of the abdomen was 6 cm. long; it had the color of the intestinal mucosa and was covered with mucus. A round opening was visible on either end of the mass, which seemed to be only slightly congested and showed no signs of inflammation. The general condition of the patient was not affected and the infant was gaining weight.

No difficulty was experienced in reducing the prolapse but tight bandaging did not prevent repeated eventrations. A few minute erosions appeared on the surface of the prolapsed mucosa. Fecal masses continued being expelled through the enterostomy as well as through the rectum but the prolapse recurred every day. Three unsuccessful attempts were made to overcome the recurrences by approximating the edges of the wound with deep silkworm sutures. The skin surrounding the stoma was highly macerated in spite of applications of latex, kaolin, etc., and the stitches would not hold. Finally, a rubber sponge was applied to the wound, which immediately stopped the recurrences of the prolapse. The surrounding skin gradually assumed a normal appearance and the enterostomy closed spontaneously.

Two and half months after the first operation the child weighed 9 lb. 2 oz. (4.61 kg.), i.e. it had gained three pounds (1.35 kg.).

DISCUSSION

The first observation which attracts attention in this case is the development of peritonitis and paralytic ileus after the Rammstedt operation, in spite of the fact that no visible perforation of the pyloric mucosa was produced. As the pus from the subphrenic abscess contained colon bacilli, it is reasonable to assume that the infection was caused not by an accidental contamination but by a minute leakage of the gastric contents through the exposed mucosa of the pylorus.

Another interesting feature is the failure of the roentgenogram to reveal a shadow indicating the subphrenic abscess, although the overlying gas bubble was visualized. It follows that in suspicious cases an exploratory aspiration or incision is justified even if the *x*-ray findings are negative.

The most important complication was the eventration of invaginated intestinal loops. Bérard and Chalier,[1] who were the first to describe a prolapse of intestines through an artificial anus, distinguish two stages of the condition: (1) eversion of the mucosa and (2) formation of a mucous cylinder. Mallet and Martin[2] reported 7 cases and ascribed the prolapse to the suction exerted by Reverdin's receptacle, worn by all their patients. Recently Jaeger[3] observed a similar condition. All cases reported in the literature developed in adults with colostomies of long standing. The mechanism of the development of such a prolapse is illustrated below (Figs. 1 and 2). A vis a tergo, i.e., an increased intra-abdominal pressure pushes the posterior wall of the attached intestinal loop in the direction of the least resistance. Gradually a cylinder forms; it has two orifices, one leading to the afferent, the other to the efferent loop. The external wall of the sausage-shaped mass is formed by the everted mucosa, the internal wall by the serosa, and the stem by the mesentery. If the pressure is sufficiently strong, the cylinder is transformed into a pouch which may serve as a hernial sac for other intestinal coils. A strangulation may develop in such a pouch. As to the pathogenesis of such combined evisceration and intussusception, the following conditions are the determining factors: (1) wide stoma; (2) relaxation or atrophy of the abdominal muscles; (3) vivid motor activity of the opened and fixed intestinal loop;

[1] Bérard L. and Chalier, A. *Lyon Chir.*, 2: 96, 1909.

[2] Mallet-Guy P. and Martin, E. *Journ. de Chir.*, 34: 425, 1929.

[3] Jaeger, F. *Bruns' Beitr. z. Klin. Chir.*, 163: 539, 1936.

(4) increased intra-abdominal pressure; and (5) long mesentery.

Contrary to other observations described in the literature, the complication developed in an infant and not in an adult; in an ileostomy and not in a colostomy; in a relatively recent stoma and not in an artificial orifice of a long standing. Pads and tight dressings, recommended by writers on this subject, proved useless in the author's case. A rubber sponge accomplished the desired effect, probably by exerting a continuous elastic pressure and absorbing moisture, thus preventing maceration and contributing to an increased strength of the abdominal wall.

Figure 1

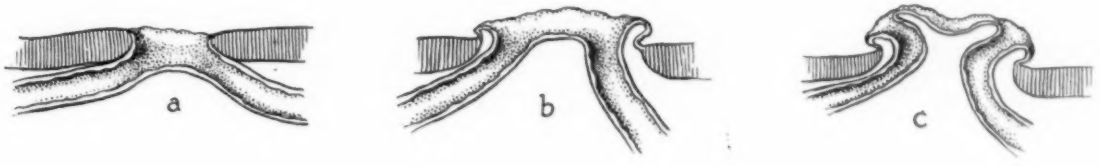

Figure 2

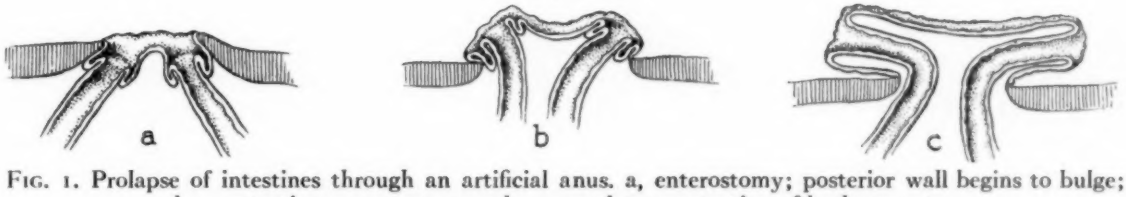

Fig. 1. Prolapse of intestines through an artificial anus. a, enterostomy; posterior wall begins to bulge; b, eventration more pronounced; c, complete eventration of both stomata.

Fig. 2. Intussusception and evisceration through an artificial anus. a, intussusception of the afferent and efferent loops; b, intussusception combined with incipient evisceration; c, intussusception and evisceration completed. Note: This mechanism results in a protrusion of a much larger loop than a simple bulging of the posterior wall of the attached gut, as illustrated in Figure 1.

SUMMARY

A Rammstedt operation for a pyloric stenosis was performed on a four weeks old infant. One week later a paralytic ileus necessitated an enterostomy. Six days after this second operation a subphrenic abscess and four days later a pelvic abscess were drained. On the top of all these complications a prolapse of invaginated afferent and efferent loops developed through the enterostomy opening. A rubber sponge applied over the stoma proved effective in the prevention of recurrences of this intussusception and eventration. A spontaneous closure of the enterostomy followed.

NEW INSTRUMENTS

MECHANICAL HOLDER FOR RETRACTORS IN NEUROSURGERY

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WORKING through skull defects of limited size upon structures often situated at considerable depth, neurosurgeons frequently employ electrically lighted retractors to obtain adequate visualization of surgical lesions. Due to the narrow field of view the assistant generally is unable to keep the lesion in view constantly so the surgeon is obliged to hold the retractor for himself. This forces him to operate with one hand while the use of both is more desirable. This is particularly necessary when the organization of the operating clinic does not provide for assisting members of the team to remain on service long enough to learn to render the most efficient assistance.

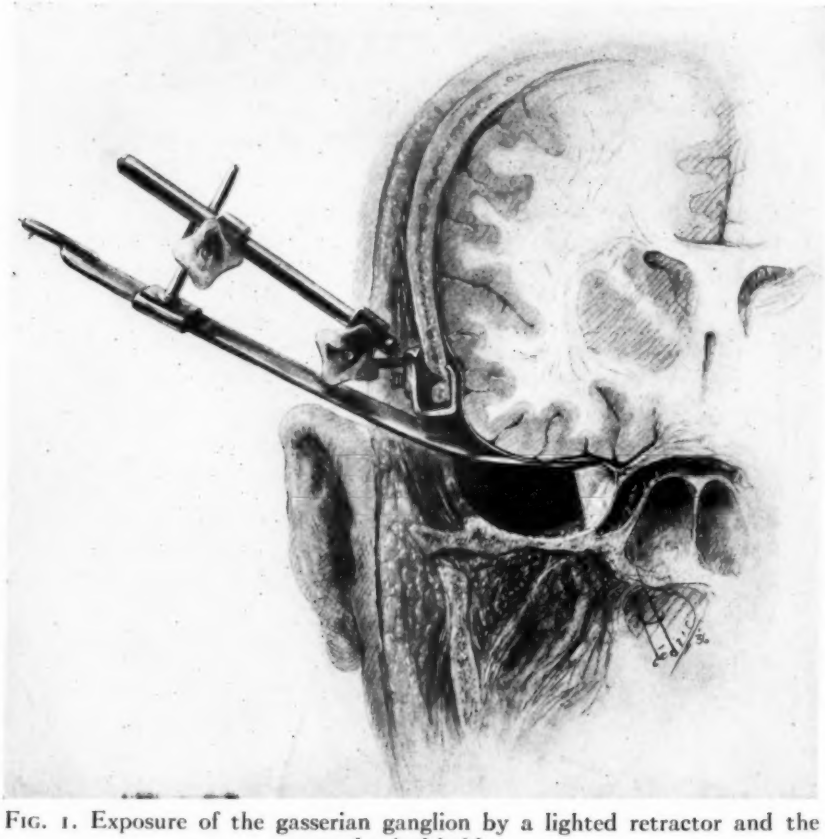

FIG. 1. Exposure of the gasserian ganglion by a lighted retractor and the mechanical holder.

In other fields of surgery self-retaining retractors have been employed to advantage. Dr. A. J. Crawford of Detroit,

Michigan, has devised an instrument made on the principle of a self-retaining orificial speculum for visualization of the gasserian ganglion. Although I have not been able to find a reference in the literature, it has come to my attention that Professor Th. de Martel of Paris has devised an efficient instrument for retaining retractors in neurosurgery.

The instrument here described was made to hold the retractors commonly used in neurosurgery, thus allowing the surgeon the use of both hands for operating. It consists of a clamp that is attached securely with a screw to the edge of the bone defect outside the dura. This remains in place throughout the operation. The blade of the lighted retractor is slipped into a slot in the holder where it is retained by friction. These two parts are joined by a connecting rod and two thumb screw joints allowing a universal range of motion. When the retractor tip is placed in the proper position, the two thumb screws are tightened by an assistant and the retractor is fixed rigidly in place, leaving the surgeon's hands free for operating. The position of the retractor is easily changed by loosening the two thumb screws and redirecting it, after which the screws again are tightened.

Originally made for exposure of the gasserian ganglion and its sensory root in trigeminal rhizotomy, the instrument has proved useful for retracting the cerebellum when removing acoustic neurinomas and for maintaining exposure of lesions in the neighborhood of the sella turcica. One or more of the retractors may be so secured when several are required for exposure of other intracranial lesions, with diminution of trauma to the cortex that sometimes results from the varying pressure exerted by the hand of an assistant. The time consumed in adjustment of the instrument is negligible in view of the excellent exposure obtained.

NEW INSTRUMENT FOR INTESTINAL ANASTOMOSIS*

PRELIMINARY REPORT OF EXPERIMENTAL WORK

FRANK GLENN, M.D.

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OVER 250 different methods of resecting segments of intestine and uniting the divided ends have been reported in the literature; had any of these been entirely satisfactory it would be superfluous to present new procedures. However, the problems of preventing trauma to the intestinal wall, contamination of the field of operation and postoperative intestinal obstruction have not been solved satisfactorily.

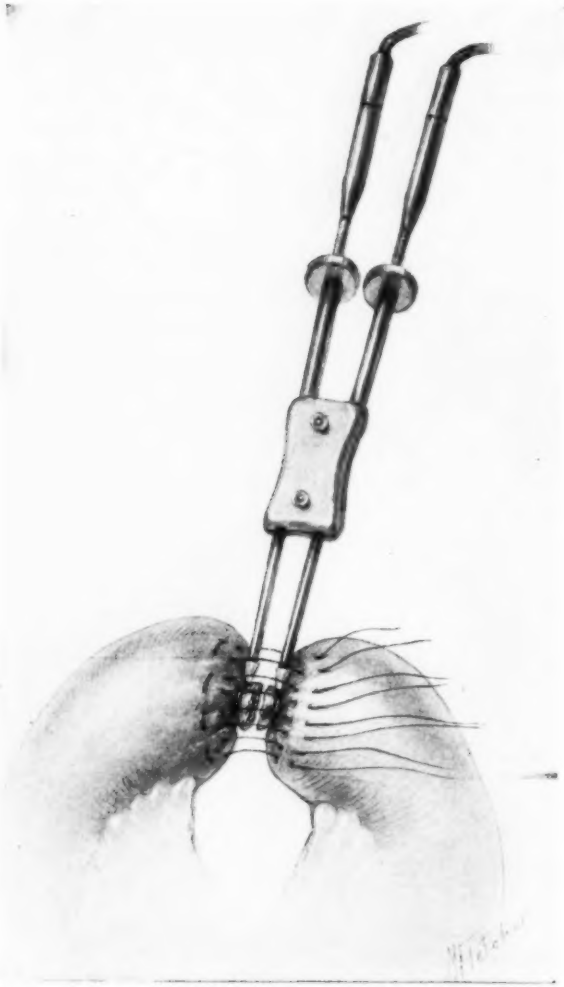

FIG. 1. The instrument is shown after the resection of a segment of bowel, with the stumps held in alinement. The bakelite tubes through which the snares operate are in position in the special crossbar clamp.

In an effort to find a means of avoiding these difficulties, experiments on end-to-end anastomoses have been performed for some years. The original experimental work was done by Halsted, who used the bulkhead principle in cutting through the inverted, closed ends of the bowel. Horine, Fraser and Dott and others worked on a simplification of Halsted's method and following the same principle, Burket and McClure continued the research which forms the basis for the study to be described briefly.

Burket and McClure employed a wire loop similar to a tonsil snare to cut through the closed stumps of bowel. The procedure necessitated the withdrawal of the snares through the line of sutures which united the intestinal ends. As the wires were withdrawn they tended to drag the submucosa with them into the metallic tube; also, the operation frequently left a wide cuff of intestinal wall within the lumen. To obviate these difficulties, a diaphragm of knife-blades and various kinds of loop sutures later were tried without complete success.

The instrument to be described is constructed on the principle of the snare employed by Burket and McClure but it circumvents some of the difficulties encountered by these authors. It consists of two wire snares with attachments to a Bovie unit. The snares operate through bakelite tubes which can be held in close alinement in a specially constructed crossbar clamp. The instrument (Fig. 1) is used in the following manner: A snare is placed

* From the Department of Surgery of the New York Hospital and Cornell Medical College.

at each extremity of the segment of bowel to be resected after the mesentery has been divided and the blood vessels ligated. By means of set-screws, the snares are tightened until they obliterate the lumen of the intestine. Using the cautery, the intestine is divided close to the snares and removed with its mesentery. The stumps of bowel held in the snares are now brought into alinement by approximating the bakelite tubes and are secured in this position by fitting the tubes into the cross-bar clamp. The divided ends of bowel are brought together with mattress sutures, one of which is placed so that it embraces the snares. Beginning with those closest to the mesentery, all sutures are tied with the exception of the one embracing the snares. The connection with the Bovie unit is made now and the current through the snares cuts cleanly through the inverted stumps of bowel, leaving within the lumen a very small amount of untraumatized intestinal wall. The instrument is now discarded and the last mattress suture tied. Reinforcing sutures are placed between the mattress sutures and the abdomen is closed in the routine manner.

In 1737 the University of Göttingen ("Georgia Augusta") was formally opened (September 17), and the Royal Medical Society of Edinburgh was founded.

In 1837 Gerhard differentiated between typhus and typhoid fevers. Colles stated the law of maternal immunity in syphilis. Jacob Henle described epithelial tissues. Rush Medical College (Chicago) was founded. K. k. Gesellschaft der Aerzte was founded at Vienna.

In 1437 Eugenius IV chartered the University of Caën. In 1137 St. Bartholomew's Hospital (London) was founded by Rahere.

In America men of great financial resources have outpaced foreign governments in liberal endowments for scientific and medical research.

From—"History of Medicine" by Fielding H. Garrison, M.D.

DEVICE FOR TIGHTENING WIRE FOR BONE FIXATION

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THERE are many situations which arise during open operations for fixation of bones where wire is the best material for holding the fragments. A notable example is where the fracture is committed near a joint and involves cancellous bone. Screws will not hold in such structure and it is not always feasible to use a bolt.

Where wire is used there is always the difficulty of tightening it by twisting without breaking it. Tightening a wire by twisting the ends together is bad practice because the wire is frequently broken or, worse, is strained to the breaking point and breaks later when stress is applied.

The device illustrated has proved very satisfactory since it was first tried. The wire is passed through the threaded stem, A, and locked by the lock nut, B, at the top. It is then tightened by running the knurled wheel, C, down the threaded stem until the resistance felt to turning indicates that the wire is tight enough. It is then twisted by turning the entire instrument and at the same time loosening the lock nut.

An advantage which the instrument possesses is that it can be used in a deep or small incision.

FIG. 1. Wire fastened in instrument and tightened. A, threaded stem; B, lock nut; C, knurled wheel.

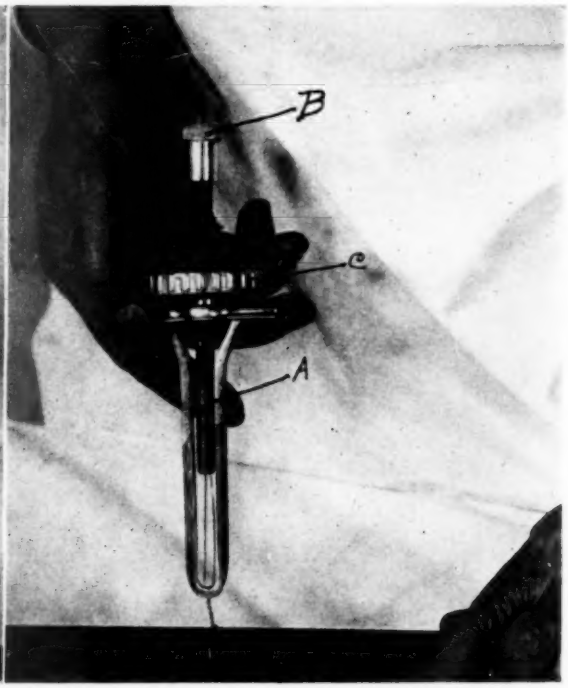

FIG. 2. Wire locked by twisting entire instrument. A, threaded stem; B, lock nut; C, knurled wheel.

A NEW SURGICAL INSTRUMENT WHICH SIMULTANEOUSLY CUTS AND RETAINS A SUTURE*

DAVID I. SOLOMON, M.D.

NEW YORK

THE conventional technique of handling retention sutures in surgery is to clamp the suture about 4 inches from the tie and then to cut off the redundant material with a scissors. This procedure requires two motions, two instruments and frequently the help of the assistant or nurse.

The instrument described herein cuts and clamps the suture with one stroke. This eliminates one motion, one instrument and the help of the assistant. It is suggested that the name Kut-Klamp be used to designate this instrument and we will refer to it as such hereafter.

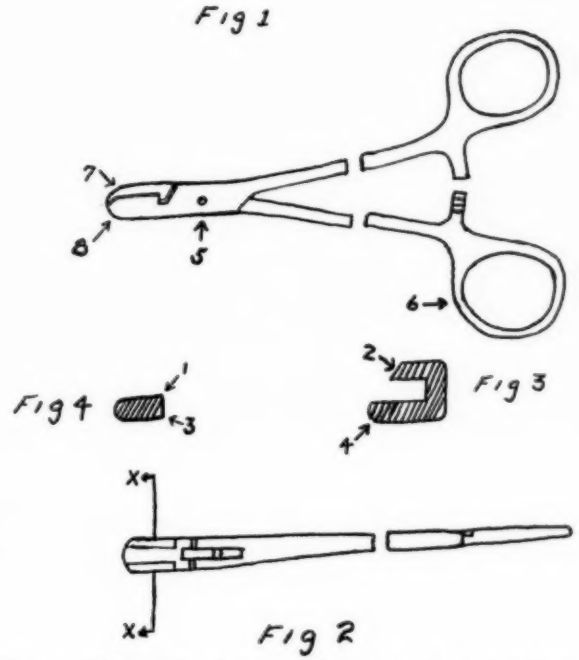

FIG. 1. Side view. FIG. 2. Edge view. FIGS. 3 AND 4. Cross section through Figure 2 at *x-x*.

The Kut-Klamp is shown in detail in the accompanying drawing.

From the lock, 5, to the end loops, 6, it is 4 inches long and is similar in design to the conventional clamp. From the lock to the tip of the instrument it is 1 inch and in it are the jaws, 7 and 8, which embody the innovation.

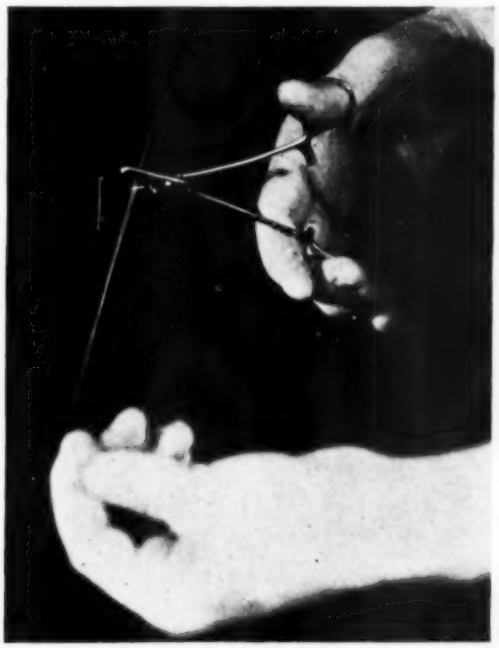

FIG. 5. Before cut.

The two jaws, 7 and 8, may be described by referring to them as male and female. As shown in Figure 3 the female jaw is U-shaped in cross section and has a groove extending lengthwise along the jaw. The male jaw (Fig. 4) fits this groove closely. As shown, one side of the male jaw, 1, is ground and sharpened to provide a cutting

*From the Gynecological Division of Harlem Hospital in New York City, Dr. Henry C. Falk, Director.

edge, and the corresponding side of the female jaw, 2, has been sharpened to provide a cutting blade. They thus function as shears. The other side of the male jaw, 3, has been left blunt, thus providing for a clamping action when it meets, 4, also blunt.

Fig. 6. After cut.

It will be noted in Figure 3 that the clamping side, 4, of the female jaw is higher than the cutting edge, 2. Thus the suture is caught and clamped before it is cut.

To indicate the cutting side the lock pin is made with a knob-like projection, 5, on this side.

In using the Kut-Klamp the surgeon picks up the instrument, the knob-like projection, 5, indicating to him the cutting side. The suture is grabbed in the Kut-Klamp and the jaws closed. With this one stroke the suture is clamped and cut, and the conventional latch arrangement retains the jaws in the closed position.

This procedure is distinctly illustrated in the accompanying photographs (Figs. 5 and 6).

This instrument is at present being used at the French and Harlem Hospitals in New York City.

[From Fernelius' Universa Medicina, Geneva, 1679.]

BOOKSHELF BROWSING

THE LIFE AND TIMES OF THOMAS SYDENHAM*

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BROOKLYN, N. Y.

IN 1924 medical men in all parts of the world foregathered in their various meeting places and celebrated the tercentenary of the birth of Thomas Sydenham. On that occasion many distinguished men delivered eulogies upon his life and work and accomplishments; tablets and busts, dedicated to his memory, were unveiled and hospitals were named for him and endowed in his name. This was really in a way a very remarkable phenomenon, not alone because of the fact that no one more than he, himself, or, for that matter, any of his contemporaries, would have been more surprised at such an action, but particularly remarkable in that this thing could not possibly have occurred at an earlier date. As I sense it, it seems to be the fate of most ordinary mortals to have a proper appreciation of their outstanding virtues delayed until immediately after their demise. Such appreciation is usual, perhaps ordinary, short lived and of little note. Sydenham not being an ordinary mortal presents a very different picture.

He grew more in stature during the sixth half century following his birth than during any other period. It required the lapse of nearly three hundred years before the profession came to realize the importance and significance of his contributions to the progress of medicine. Surely such a man warrants our consideration.

We can perhaps get a better picture of Sydenham if we pause for just a moment and consider very briefly the times immediately preceding his birth and during which he lived. These times were crowded with important events and perhaps had greater influence upon succeeding events than any other similar period of history.

Elizabeth had completed her long and notable reign. Spain had been eliminated, apparently forever, as an important world power. Literary excellence had attained an all time high water mark in England and other parts of Europe, notably France. Shakespeare had wined and dined, perhaps wisely, undoubtedly at times too well with his cronies Marlowe, Ben Johnson and others. Between bowls of wine, a good story and perhaps a song or two he had scribbled bits of verse on theatre programs and other odds and ends of paper and these fragments, without further editing or embellishment were the sonnets which we read and admire today. In a brief period of less than twenty years he had written his

* Read before a meeting of the Brooklyn Hospital Staff, January, 1936.

immortal plays and retired to Stratford to enjoy the last few years of his relatively short life as a retired gentleman of means. A little later Milton was pondering over his great epic poem and finally, out of the total darkness of his own life, dictated Paradise Lost to his devoted daughters. Locke was preparing to write his philosophical essays, Pepys was writing his diary and many other writers were contributing their quota to the classics of today.

The Holy See at Rome, after years of meddling in European politics had, in the opinion of many, become corrupt. Luther in Germany and later John Knox in Scotland had dared to rise up and not only challenge the infallibility of the Pope but had further dared to challenge his interpretation of the Scripture and to refute the theological and ritualistic dogmas which had carried down through the ages. The peace of Europe was being constantly threatened by differences of religious opinion. Witches were burned at the stake and wholesale massacres were not uncommon. People took their religion seriously in those days. They spent their lifetime savings for the erection of mighty cathedrals, whose architectural beauty and magnificence have never been surpassed; they were willing and they did shed their blood for their beliefs. May I say in passing that both the proponents and the protestants have, since those olden days, come staggering down through the centuries. They have long since ceased to murder one another but each in their own way have, in my humble opinion, drifted and are continuing to drift further and further away from the very simple but very profound philosophy propounded nineteen hundred years ago by the carpenter of Nazareth.

Elizabeth was succeeded by James, the son of her cousin, the ill-fated Mary, Queen of Scots. His elevation to the throne firmly united the Kingdoms of England and Scotland and might perhaps be interpreted as the beginning of Great Britain. His one other claim to fame lies in the fact that his name is attached to the Bible as we know it today. His relatively short reign was followed by that of his son, Charles I. Charles undertook to establish himself as an absolute monarch and lost his head in the effort. There followed the interregnum of Cromwell, whose cause was staunchly supported by Sydenham. Persecutions in the name of religion continued and were very definitely a factor in speeding up the colonization of our own country. The colonies were becoming organized; John Harvard was starting his little school in Cambridge and the decimation and eventual annihilation of the American Indian was getting under way.

London had become a great metropolis with a population of half a million. Culture and education among the gentry had reached a high peak. Latin was an outstanding feature in the curricula of all schools and colleges and no gentleman's education was considered complete until he had acquired a knowledge of this language which would enable him to read and to write it quite as fluently as his native tongue. These were the times and this the atmosphere into which Sydenham was born and through which he lived. His life span carried through the periods of Charles I, Cromwell and Charles II, who was recalled to the throne from his exile in France, following the decline of Cromwell. Compared to the turbulent times immediately preceding, the reign of Charles II was relatively peaceful and uneventful.

While all of these tremendously important events were transpiring in the world at large, events of equal importance had occurred and were occurring in the development of medicine and surgery.

After the lapse of many centuries, during which the teachings of Galen and the Arabian school had been blindly followed and few if any had thought of questioning them, Vesalius had dared to challenge their authority. He had made his careful painstaking dissections, created the science of anatomy and had written one of the great, perhaps the greatest medical classic of all time. Other brilliant anatomists, Sylvius,

Fallopius, Willis, Steno and many others, getting their inspiration from the great master, had supplemented his work.

Ambrose Paré had rescued surgery from the hands of the barber and elevated it to the rank of a science and an art.

Jansen had experimented with the simple lens of Roger Bacon, DaVinci and Gallileo and produced a microscope. With its aid Malpighi had delved into the ultimate stricture of the body tissue and established histology as a science. Von Leeuwenhoek further improved the microscope and finally produced an instrument not unlike what we use today. He saw and described and made drawings of bacteria and, strange to relate, this man, the janitor of a district court house, not only did all of this but he was the first man who actually saw the blood cells coursing through the capillaries. This phenomenon he described accurately and thus provided the one missing link in the epoch making discovery of Harvey reported a few years before. Harvey made his monumental contribution during the early days of Sydenham's life. The circulation of the blood had been suggested many years before by several writers; Aristotle, Galen, DaVinci and others; and Servetus had already written an accurate description of the pulmonary circulation. However to William Harvey belongs the credit of this great and epoch making discovery.

Time will not permit of any further elaboration of this phase of my topic but enough has been said to demonstrate that scientific medicine was getting under way when Sydenham came upon the scene.

However, it is only fair to say also that these data had not been assimilated nor had their significance been sensed even in a small degree. The practice of medicine was still stultified by the traditions of the centuries and the teachings of Galen and his many disciples. It was based in large measure upon a combination of more or less weird conceptions of anatomy and physiology, together with many equally weird superstitions. The left hind leg of a rabbit killed in a graveyard during a full moon was still a sovereign remedy for many ills.

Sydenham never got away entirely from this atmosphere. He apparently attached little significance to Harvey's contribution, at least he never mentioned either Harvey or his work in his voluminous writings. Of course he must have known about them as he and Harvey were in a sense contemporaries. In fact, for all we know, they may have been neighbors or even personal friends. On the other hand, he was not tethered by tradition. He believed only what he saw himself and made his deductions and conclusions from personal observations made at the bedside. He was undoubtedly the first great clinician since Hippocrates.

Sydenham was born in Dorsetshire in 1624, just four years before Harvey had demonstrated the circulation of the blood. He was the son of a moderately well-to-do and genteel family and his education was planned and carried forward along lines befitting his station in life. He eventually enrolled as a student at Oxford but left shortly after his enrollment to take up arms with Cromwell against the King. During the interregnum he returned to Oxford and graduated with the degree of Bachelor of Medicine. Sometime in his thirties he settled in London, started in practice and at the age of thirty-nine became a Licentiate of The Royal College of Physicians but was never admitted to fellowship in this august body. However his bust occupies a place of honor in front of their building today. He was doubtless little if at all disturbed by this attitude. Such honors had little appeal for him. He said in a letter to a friend, "I have weighed in a nice scrupulous balance whether it is better to serve men or to be praised by them, and I prefer the former." He did, however, receive a doctor's degree from Cambridge when he was fifty-two. Sometime during this period, probably fairly early in his professional career, he spent some time in France working with the great French clinician, Bar-

beirac. The methods, thoughts and activities of these two men had many points in common and they have often been referred to as the French and the British Hippocrates.

A popular conception of the nature of disease during the early days of Sydenham was the chemical one. It was assumed by many that all diseases were due to differing types of fermentation, which produced either an excess of acid or an excess of alkali and that the proper treatment depended entirely upon recognizing upon which side the error lay and administering acids or alkalis accordingly. Of course this seems a bit fanciful and yet we of today talk glibly about acidosis and alkalosis, perhaps with good reason, and yet I wonder what the practitioners of the twenty-third century will think about it.

Sydenham was not particularly interested in the fundamental nature of disease. He was rather interested in a recognition of the symptoms, the course it might be expected to take, its incidence in reference to locality, seasons, climatic conditions, etc., whether or not it was likely to occur as an isolated instance or spread in epidemic form, and methods of relieving symptoms and effecting a cure. He summed up his own conception of disease processes in general in rather a shrewd definition. He said, "Disease is an effort of Nature, striving with all her might to restore the patient by elimination of the morbific matter." In the language of our friend Al Smith, "Think it over." I think it is a pretty good definition even today.

His description of many diseases, smallpox, pulmonary tuberculosis, chorea, gout, malaria, hysteria are classic and well worth reading even today. Chorea still bears his name. He described accurately the quotidian and tertian type of malaria. He demonstrated that hysteria as a symptom complex was not confined to the female sex. He wrote very feelingly of gout since he was a victim himself. He also apparently suffered from urinary lithiasis since he describes frequent attacks of hematuria and gravel. His methods of treating these symptoms, omitting surgical measures about which he of course knew nothing, was highly intelligent even when judged by the standards of today.

His description of pulmonary tuberculosis or phthisis, as he called it, is well worth quoting. He said,

> The cough betrays itself. The phthisis comes on between the ages of eighteen and thirty-five years. The whole body becomes emaciated. There is a troublesome hectic cough, which is increased by taking food and which is distinguished by the quickness of the pulse and the redness of the cheeks. The matter spit up by the cough is bloody or purulent. When burnt, it smells fetid. When thrown into water it sinks. Night sweats supervene. At length the cheeks grow livid, the face pale, the nose sharp. The temples sink, the nails curve inward, the hair falls off and there is a colliquative diarrhea, the forerunner of death.

One could hardly ask for a more vivid description. It shows very keen powers of observation and must have represented many hours of study at the bedside of his patients in their homes. (Remember that, throughout his life he never had a hospital service.) It seems strange that so keen a man did not go one step further and percuss or put his ear to the chest of his patients. Apparently he never did or, if he did he never wrote about it.

During the active period of Sydenham's career, a severe epidemic of bubonic plague spread over London with an enormous loss of life. During most of the time that this epidemic was raging over the city, Sydenham was away in the country. Some of his biographers have criticized him for this apparently cowardly attitude. In his defense we should perhaps consider that after all he was engaged in private practice mostly among the well-to-do, that he had no hospital affiliations and that most of his patients had also fled the city. He did however return before the epidemic was over, saw a few cases and commented very shrewdly upon them, particularly in connection with the manner of spreading. It is rather interesting to note in passing that

various observers of those days suspected the dog and the cat as carriers. They just missed the rat; another one of the many instances in which an important fact has been missed by the narrowest of margins.

Perhaps his greatest contribution to the literature was his work on fevers and fever in general. This work was revolutionary and was not only far ahead of his own times but apparently far ahead of the times of many succeeding generations. He maintained that fever was a wholesome process and was Nature's own method of combatting noxious substances in the system. It required the lapse of two hundred and fifty years before the profession could assimilate this simple but important fact. No longer ago than the early years of the present century men were needlessly combating fever with phenacetin, acetanilid and various other so-called febrifuges.

His therapeutic measures were, on the whole, excellent even when judged by present day standards. He was the first man who had the courage to rely occasionally on a course of masterly inactivity and give Nature a chance to effect a cure. He was firmly convinced that no remedies should be applied unless clearly indicated. He stressed the importance of Nature's own remedies, fresh air, sunshine, plenty of water to drink, dietary regulation and other hygienic measures. Particularly in fevers did he advocate fresh air and an abundance of cold water and other cooling drinks. This, too, was revolutionary and again required a lapse of two hundred and fifty years before it was accepted. I well remember when as a youngster I had measles, I was placed in a hot stuffy room, with windows tightly closed and plied with hot nauseating drinks, all for the purpose of bringing out the rash. I would have given anything which I ever hoped to possess for one drink of cold water. I believe that was the standard treatment of those days. Of course I had never heard of Sydenham then. I am equally certain that my attending physician had never heard of him either.

Of course Sydenham did use drugs; even today laudanum is spoken of as Sydenham's tincture. He employed venesection, which was very popular in those days. He even resorted occasionally to ridiculous decoctions containing a multitude of ingredients, one in particular which was said to contain something like eighty ingredients and required three months for its preparation. As I said a little earlier, he never got away entirely from the traditions and superstitions of preceding ages.

As one reflects upon the career and activities of Thomas Sydenham as a practicing physician, I wonder if we, even of today, cannot discern a lesson. Surrounded as we are and perhaps a bit spoiled by an endless list of technical aids, are we not prone to forget that we too have eyes and ears and fingers, perhaps a bit of common sense and in the course of time, a modicum of experience?

While, of course, there is no reason in the world why all or any of our modern aids to diagnosis should be thrown out of the window (three letters at a time or any other way) I do think that on many occasions we might, to the mutual advantage of our patients and ourselves, invoke much more freely than we do the very simple armamentarium which served Sydenham so well.

Sydenham had very few pupils. Perhaps the most notable among those few was Dover, of Dover's powder fame. In this connection, remember again that he had no hospital affiliations and therefore was not geared for teaching. What teaching he did was largely through the medium of his writing and he wrote extensively, always in Latin, according to the custom of scholars of his times.

His papers and books were published in practically all of the countries in Europe and were eventually translated into English, gathered in one volume and published in America. Every bit of his writings were based absolutely and entirely on his own personal clinical observations. They were concise, to the point, carried no excess

verbiage and never referred to previous authorities. Perhaps he erred a bit in that. On the other hand, I wonder if this, too, does not provide a thought for the writers of the present day. It would seem that no paper of today is complete without two or three pages of bibliography. Of course this is perhaps commendable and often essential in certain types of papers and yet I have a feeling that it is a bit overdone. After all, isn't the best paper the one that contains some one single original thought, briefly and concisely presented? One of the greatest scientific papers of all time, Einstein's original paper, occupied half a page in a scientific journal.

One might ramble on almost indefinitely discussing many other phases of this interesting period and the influence which Sydenham had upon it. However, I fear that I am exceeding my time allowance.

May I conclude by saying that, in my opinion, Sydenham does not quite belong in the very small group of intellectual giants who grace the pages of medical history. He does not, either by his attainments, by his accomplishments or by his influence upon the subsequent course of medical and surgical progress, quite measure up to the stature of Hippocrates, Galen, his own contemporary Harvey, John Hunter, or in more modern times, Virchow and Pasteur, and a few others who might be included. He does, however, occupy a distinguished position in a slightly larger group and his name will for all time be accorded a place of honor in the blue book of medical and surgical history.

BOOK REVIEWS

Recent Advances in Laryngology and Otology. By R. Scott Stevenson. Philadelphia, P. Blakiston's Son & Co., Inc.

This volume on recent advances in otolaryngology is very well conceived and put together by the author. In his preface he recommends the volume to students and house surgeons in otolaryngology and very timidly suggests that the volume also might be of value to more advanced practitioners in the specialty. In the opinion of the reviewer the volume most certainly is of great advantage to the practitioner of otolaryngology because it gives, in concise and clear language, a review of the advances made in each section of the specialty over a period of the last five years. The advances are in many places very critically reviewed by the author in the light of his own experience, and, if for no other reason than these critical reviews the volume would be of great interest to the average practitioner. There is also in the references at the end of each chapter something which is of very great advantage to the specialist who wishes to carry his study further by a personal review of the literature studied by the author. From the reviewer's point of view this is also a very valuable and very important contribution to the education of the busy specialist.

This book is also commended for study to those American students of otolaryngology who are preparing themselves for the examination by the American Board of Otolaryngology, because it gives to them in a very small volume many of the pathological and physiological studies, searching for which they would have to spend a great deal of time and energy studying the recent periodicals.

It is hoped that when the next five years has elapsed another volume written in the same style will be available.

Aural Therapy in Relation to Deafness. By Prof. D. F. Fraser-Harris. London, Sterling Medical Publishing Co.

This is a small brochure apparently written for the general practitioner and the laity bringing forth the uses of the audiometer in the study of deafness and attempting to describe in simple terms some of the types of deafness in relationship to audiometer curves. The reviewer can see no reason for the chapter on treatment with the author's thermocatheter. The few lines on the use of hearing aids perhaps could have been done with greater thoroughness.

Textbook of General Surgery. By Warren H. Cole, M.D., F.A.C.S. and Robert Elman, M.D. New York, D. Appleton-Century Company, 1936. Price $10.

We learn that this volume of 1031 pages is a direct outgrowth of a formal recitation and lecture course in surgery given by the authors to the third year class at the Washington University School of Medicine during the past eight years. In the Preface we read, "Most of the text, indeed, represents the teaching material resulting from the use of mimeographed assignments which have been repeatedly revised and corrected during the past three years. . . . The text also includes the more advanced details, which fourth year students are expected to know before graduation. . . . The material included should also suffice for the every day use of the general practitioner." The authors also strike a pleasing note when they say, "If more extensive details of a particular surgical disease are desired, a system of Surgery will be required; it is obviously inappropriate to include a complete discussion of one or more diseases without doing so for all, and this is impossible in a single volume work."

With this in mind, the authors have eliminated detailed consideration of the specialities, ophthalmology, otology and rhinology. On the other hand, they have stressed such allied specialities as genitourinary surgery and gynecology. Certain details of neurosurgery, especially head injuries, have been included.

The authors have stressed the subject matter from a physiological angle, for, in this way the clinical features will be under-

stood better. More detail of case histories in the legends under the illustration wherever possible has been the rule.

The chapter on the emergency prostrate patient, which deals more or less with the unconscious individual should prove of value to the interne, general practitioner and young surgeon. There is a tendency to insist that a textbook is mainly of value to an interne or the young man just beginning surgery. It is our opinion that many middle aged and past middle aged men, and many more or less well known workers in the surgical field may spend no inconsiderable time with modern textbooks relating to their sphere of work. Therefore, this volume being exceptionally good, we suggest to medical teachers of surgery to hint to their students that they might do well, if possible, to own copies.

There are 559 figures, a comprehensive Bibliography at the conclusion of each chapter, and an Index.

THE EYE AND ITS DISEASES. By 82 International Authorities. Edited by Conrad Berens, M.D., Ophthalmic Surgeon, Pathologist and Director of Research, New York Eye and Ear Infirmary; 1254 pages with 436 illustrations, some in colors. Philadelphia and London, W. B. Saunders Company, 1936. Cloth, Price $12.00 net.

This textbook of ophthalmology, edited by Dr. Berens, is an excellent, authoritative book and a valuable addition to the literature of the speciality. Dr. Berens feels that ophthalmology, in view of the constant increase in our knowledge of the eye, has reached the stage where it is beyond the power of one individual to write authoritatively on all its varied phases; and because of this the ideal text should consist of the expert knowledge of many oculists, each writing of the subject in which he is an acknowledged authority. With this in mind, Dr. Berens has searched the United States, Canada, England and the Continent for contributions to this book and requested opinions on their particular assignment from those he thinks best qualified. While many will feel that this is carrying specialization in medicine too far, the results in this book speak against such a conviction, because in the not too well supplied field of English ophthalmological textbooks, this is one of the best.

The subject of ophthalmology is covered very thoroughly beginning with anatomy and including physiology, bacteriology, refraction, diseases of the eye and surgery; and there are also interesting chapters on hygiene, immunology, medical jurisprudence, pathological and bacteriological technique. It is a great compliment to the editor that he has been able to obtain the collaboration of such well known names in ophthalmology.

There are some defects in the book, because, as would be expected, each individual contribution represents the opinionated conclusions of its author and because of human frailties, known and accepted modes of therapy are often completely disregarded or disparaged, and this of necessity is something which should not happen in a book which endeavors to cover adequately, authoritatively and completely the subject of ophthalmology.

In view of the general excellence of the book it seems picayune to mention any such defects. As seems to happen in all textbooks, the authors are often reluctant to omit beliefs which have become traditional and are carried from text to text as a matter of habit, and we could disperse with many of them. Also accepted forms of medical and surgery therapy are often not mentioned or are lightly dismissed as of no value and those who acquire their ophthalmological information from this book will be ignorant of many definitely valuable therapeutic procedures. This should not be considered as a too severe condemnation of the book, for it is usually so good that one would like to see it even better.

Despite the great number (82) of contributors, the editor has so arranged the subject matter and the varied styles of writing, that one would suspect the book to be the work of one author. The arrangement of the subject matter, the illustrations and the type used, and the index are excellent. The book is recommended to ophthalmologists and those medical men who wish a book which brings ophthalmology up to date with current knowledge.

A TEXTBOOK OF SURGERY. By John Homans, M.D. Compiled from lectures and other writings of members of the Surgical Department of the Harvard Medical School. Fourth Edition. With a special

bibliographical index and with illustrations by Willard C. Shepard. Springfield, Ill., Charles C. Thomas, 1936. Price $8. postpaid.

The first edition of this work appeared in 1931, the second in the following year, the third in 1935, and now a year later, a fourth edition is offered to the profession. Under the editorship of Dr. Homans who made the volume possible are, Arthur W. Allen, David Cheever, Edward D. Churchill, Harvey Cushing, Robert B. Greenough, Gilbert Horrax, Daniel Fiske Jones, William E. Ladd, George A. Leland, Howard A. Lothrop, Richard H. Miller, Edward H. Nichols, Robert B. Osgood, Charles Allen Porter, Tracy J. Putnam, Lyman G. Richards, Edward P. Richardson, Channing C. Simmons, J. Herbert Waite, WymanWhittemore, and Philip D. Wilson. This work not only represents collection of outstanding authors, but it is a volume whose contents more than lives up to the claims made for it.

In this edition has been added a chapter on "Amputations and Plastics," old material has been replaced with new (the stale with the fresh), and while little has been actually added the pagination is unchanged.

The book is 1267 pages long, and contains 530 figures. The Bibliographical Index is interesting and has been especially well done. Other authors might look this over and add it to their forthcoming books.

The owner of this book will find himself, at times, putting aside the current best-seller in fiction or biography and returning to this Textbook of Surgery, for, it reads more like some human document than a cut and dried book of science.

Diseases of the Coronary Arteries and Cardiac Pain. Edited by Robert L. Levy, M.D. Advisary Editorial Committee Alfred E. Cohn, James B. Herrick and Carl J. Wiggers. New York, The Macmillan Company, 1936. Price $6.00.

During the past two decades great advances have been made in the study of the affections of the coronary arteries and of cardiac pain. Because of these advances in the knowledge of these conditions many once doomed to a speedy death are, today, offered hope of added years of usefulness.

Therefore, it is timely that this book is presented to the medical profession. Naturally, as this subject involves so many aspects, no one man could write a book on the subject and do all aspects justice. The editor, aided by an advisary editorial committee, selected fourteen men specially equipped to write on their given topics. These contributors are, Claude S. Beck, Herrman L. Blumgart, Alfred E. Cohn, Louis I. Dublin, James B. Herrick, William J. Kerr, Fred M. Smith, William C. Von Glahn, Joseph T. Wearn, James C. White, Paul D. White, Carl J. Wiggers and Frank N. Wilson.

The Preface is by Robert L. Levy, the Introduction by Alfred E. Cohn, and the Historical Note by James B. Herrick.

Part 1 deals with The Coronary Circulation (four chapters are devoted to this); Part 2 deals with Cardiac Pain (two chapters); Part 3 with Clinical Features of Diseases of the Coronary Arteries and Cardiac Pain (six chapters); Part 4 with Medical Treatment of Diseases of the Coronary Arteries and Cardiac Pain (two chapters); and Part 5 deals with Surgical Treatment of Diseases of the Coronary Arteries and Cardiac Pain (three chapters).

This excellent book of 445 pages, with an ample Bibliography at the end of each chapter, with a thorough Index, profusely illustrated, fills a needed want in the present day literature on this vital topic.

Textbook of Surgical Nursing. By Manelva Wylie Keller, B.S., R.N. Third Edition. Completely revised and reset. New York, The Macmillan Company, 1936. Price $3.

Physicians on hospital staffs who instruct nurses know this book as it is a standard work. Because this work is so widely known and used in so many institutions where nursing is taught it is unnecessary to go into detail regarding the contents.

In this third edition, the book has been rewritten in parts, revised throughout, and reset.

It is illustrated and at the end of each chapter is a list of questions on the matter covered in that chapter for student review.

Any physician or head of a hospital nursing school will feel secure in recommending this book as a textbook for student nurses.

PRACTICAL CLINICAL GYNECOLOGY

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FOURTH INSTALLMENT

CONTENTS

[*This Issue*]

Chapter		Vol. xxxvi Page
vi.	Retrodisplacements of the Uterus	750
vii.	Menstruation and Its Disorders	766

[*Previously Issued*]

		Vol. xxxv
i.	Examination of the Patient	153
ii.	Preoperative Hospital Routine; Preparation of the Patient; Postoperative Hospital Routine	172
iii.	Abortion	187
iv.	Gonorrheal Infection of the Tubes	629
		Vol. xxxvi
v.	Fibroids	559

Chapter VI

RETRODISPLACEMENTS OF THE UTERUS

The normal uterus is a freely movable organ suspended in the pelvis by the coordinated action of its supporting structures which consist of:

1. The uteropelvic ligaments running outward from the supravaginal portion of the cervix to the pelvic fascia in the base of each broad ligament. These ligaments prevent the uterus from undergoing lateral malpositions and also prevent prolapse;

2. The broad ligaments, which steady the uterus in or near the median section of the pelvis;

3. The uterosacral ligaments which pull the cervix backward and upward thereby throwing the fundus forward;

4. The round ligaments, which act as "guy ropes" and to some extent prevent the uterus from falling backward.

In the erect posture the uterus normally lies in a horizontal position with the cervix pointing backward. It is maintained in this position largely by the intra-abdominal pressure which is exerted on the posterior wall of the uterus. When the supporting structures become weakened, the anterior wall of the fundus becomes separated from the bladder and the intestines can then enter the vesicouterine space. This causes the intra-abdominal pressure to be exerted on the anterior wall of the uterus and thus gradually forces the fundus into the cul-de-sac. When the fundus is pushed backward, the cervix is forced forward with a corresponding lengthening of the uterosacral ligaments.

Pathology

What are the pathological changes produced in the uterus and ovaries as a result of retrodisplacement?

When the uterus is in a posterior position the broad ligaments become partially twisted and as a result there is an

interference with the return blood supply. This produces chronic congestion of the uterus and occasionally varicosities of the veins in the broad ligament. The retrodisplaced uterus causes a prolapse of the ovaries, which may become painful and enlarged as a result of interference with their circulation. When a postpartum or postabortal uterus is in a posterior position it does not involute properly.

Symptoms

There is a large group of patients with uncomplicated retroflexion presenting no symptoms whatsoever.

There is another group of patients where the retrodisplacement is associated with other pathology and the symptoms produced by the associated pathology completely mask any symptoms the retroflexion may cause.

Most of the symptoms of the uncomplicated retroflexion are caused by the circulatory changes—dragging sensation, feeling of weight in the pelvis, menorrhagia, backache and leucorrhea. Indefinite reflex symptoms, "loss of pep," etc., are sometimes attributed to this condition. Sterility may be caused by a retrodisplacement of the uterus due to an angulation of the tubes. Recurrent abortions may result from the impaction of a retroflexed pregnant uterus. Prolapse of the ovary associated with retroflexion is a frequent cause of dyspareunia.

Treatment

Patients with retrodisplacements of the uterus may be classified into three groups:

1. Those who are symptom free, and therefore require no treatment;

2. Those with symptoms, with replaceable retrodisplacements where the judicious use of a properly fitting pessary will relieve or cure the condition;

3. Retrodisplacements requiring relief, but which cannot be cured by nonoperative treatment.

Treatment of the noncomplicated retrodisplaced uterus producing symptoms:

Prophylaxis. Prevent the postpartum uterus from tipping backward until the relaxed supports involute sufficiently to hold the uterus forward so that the intra-abdominal pressure

A B C

FIG. 81. A. Correct knee chest posture. Chest on couch, bed or floor, knees apart, thighs up and down. After taking posture external parts are drawn open to allow air to enter. It escapes on return to upright posture. B. Incorrect. Chest on pillows, thighs slant away from body. C. Incorrect. Resting on elbows, thighs slant inward toward body.

can be exerted on the posterior surface of the uterus and thus hold it in the normal anterior position. A postpartum uterus will not fall into the cul-de-sac until after the tenth day. Why? Because it is not small enough to drop below the promintory of the sacrum. After the tenth day, patients should be encouraged to lie on their sides or on the abdomen until they present themselves at the obstetrician's office for a check-up examination. If at this time a retrodisplacement is found it should be replaced manually and a pessary of the proper size inserted. The patient should then be instructed in the proper use of the knee-chest position (Fig. 81) to help keep the uterus forward.

When the cervix is drawn forward in doing a curettage the fundus goes backward, therefore, following a curettage the uterus should always be replaced manually in its normal anterior position.

Replacement of Movable Retroflexion. The patient should be comfortably placed in the lithotomy position on an examining table. The rectum and bladder should be empty. The fingers of the left hand, well lubricated, should be introduced into the posterior cul-de-sac of the vagina and the uterus pushed upward (Fig. 82). As the uterus rises the fingers of the right hand on the abdomen attempt to catch the posterior wall of the uterus; at the same time the fingers in the vagina shift to the front of the cervix. The anterior lip of the cervix is then forced backward (Fig. 83). As the cervix goes backward the

fundus is grasped by the abdominal hand and brought well forward (Fig. 84). The pessary can then be introduced. Occasionally if the fingers are not long enough a tenaculum is placed

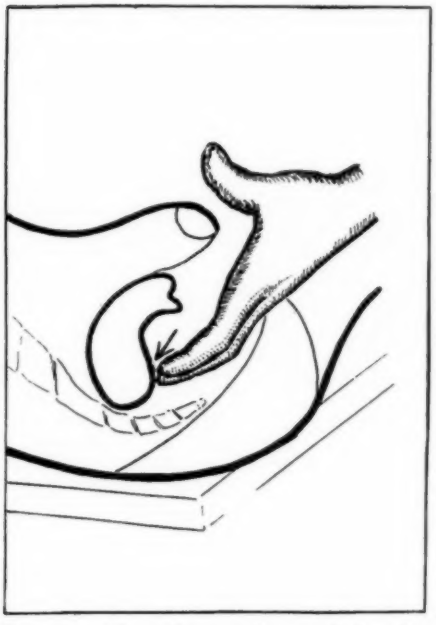

FIG. 82. Two fingers inserted in the vagina or the index finger in the vagina and the middle in the rectum, push the fundus upward in an attempt to dislodge it from the hollow of the sacrum.

FIG. 83. Two fingers in the vagina pushing the anterior lip of the cervix backward while the fingers of the other hand attempt to grasp the fundus to help bring it forward.

on the anterior lip to give added leverage. There are many other ways to replace a movable retroflexed uterus, two of which will be described.

1. This method is often used. Insert a fairly large pessary into the vagina. Place the patient in the knee-chest position and have her cough several times. This will frequently cause the uterus to fall forward. The large pessary is then removed and the proper size pessary is inserted.

2. Place the patient in the knee-chest position, insert a Sims' speculum and grasp the anterior lip of the cervix with a tenaculum. Draw the anterior lip of the cervix downward and then push it upward and backward. This may cause the fundus

to fall forward. Frequently a sponge on a stick is used instead of a tenaculum.

Function of the Pessary. The pessary does not hold the

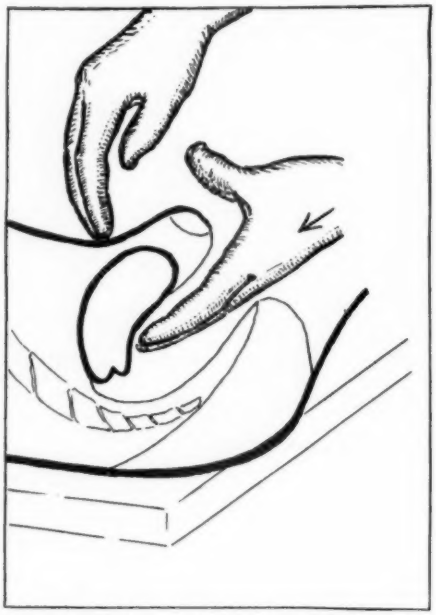

FIG. 84. The vaginal hand forcing the cervix well back while the abdominal hand pushes the fundus forward.

uterus forward, it holds the cervix back so as to allow the normal intra-abdominal dynamics to function. The pessary holds the cervix back by pushing the posterior vaginal vault backward, this throws the fundus forward, thus allowing the intra-abdominal pressure to be exerted on the posterior wall of the uterus.

Size and Shape of the Pessary. The following factors govern the size of pessary used. It should be large enough to hold the cervix well back and yet it should not project beyond the introitus. It should not cause undue pressure on any spot in the vagina. It should not be too small so as to move too freely in the vagina. It should cause neither pain nor discomfort. The patient should not be cognizant of its presence.

The shape of the pessary usually used is the Albert Smith or Hodge pessary. All sizes should be kept on hand so that they can be tried until the proper size pessary has been inserted.

Technique of Inserting the Pessary. The patient is in the lithotomy position, the pessary is clean and well lubricated. It is held by the anterior end in the fingers of the right hand

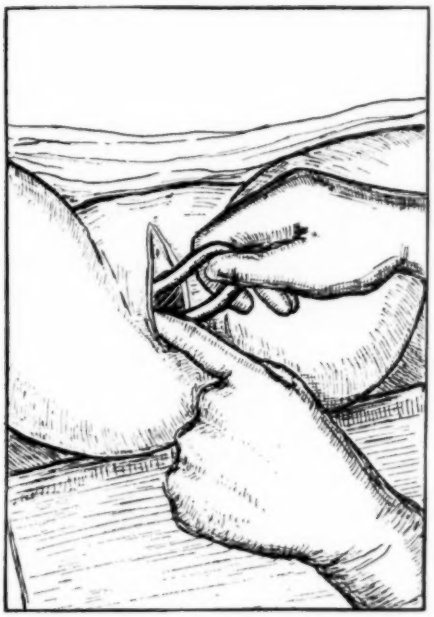

FIG. 85. Index finger of the left hand depressing the perineum. Pessary inserted obliquely by the right hand.

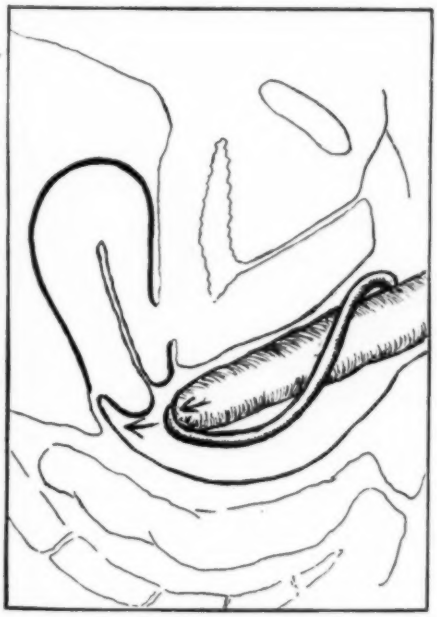

FIG. 86. Index finger on the posterior bar of the pessary depressing it below the posterior lip of the cervix.

with the large concavity slightly upward. The index finger of the left hand is introduced into the vagina and the perineum depressed. The pessary is introduced obliquely (Fig. 85). Any pressure that is exerted should be towards the perineum as the vestibule and urethra are very sensitive. When the pessary has been introduced beyond its widest part it is rotated into the transverse diameter with the large concavity upward. The index finger of the right hand is then placed upon the posterior bar of the pessary (Fig. 86) depressing this bar so as to bring it behind the cervix and to allow the cervix to lie in the opening of the pessary (Fig. 91).

The finger should then be introduced around the pessary to make sure there are no pressure spots and the patient is allowed to walk around the examining room, sit down, strain, etc., to be sure the pessary is comfortable.

Patients are frequently found whose introitus is so small that the rigid pessaries cannot be introduced. Findley has developed a pessary which has given excellent results.

With the advent of episotomies and more accurate repairs of lacerations of the pelvic floor, the insertion of these rigid pessaries, became more difficult and caused much pain to the patient. The same difficulty was encountered in treating virginal (or nulliparous) cases.

The caliber of the introitus has no proportional relationship to the size of the pessary needed for uterine support. A two-finger, recently repaired introitus will not easily admit a No. 4 or 5 Smith or Hodge pessary. A smaller pessary will often fail to hold the uterus in the anterior position. As a result, both patient and physician were prone to postpone the use of a pessary beyond the time when the best results could be obtained.

To overcome this difficulty, I have devised a modification of the Albert Smith pessary which was described in a previous publication. This instrument is identical with the Albert Smith type except that the end sectors have been replaced by soft solid rubber. These soft rubber segments are seamlessly vulcanized to the lateral arms which are of hard rubber. This arrangement allows the lateral arms to fold together so that any size can be easily introduced through a one-finger introitus. The patient frequently does not realize that the pessary has been inserted as it provokes no more sensation than that of one finger. This instrument unfolds automatically in the vagina and because of its longitudinal rigidity effectually pushes the posterior fornix upwards and backwards. This puts the uterosacral ligaments on tension and pulls the cervix upwards and backwards thus holding the uterus in anterior position.

To further simplify the application of this pessary, I fold it in the manner shown in the illustrations. As shown in Figure 87 the device is held in the position which it is to take in the vagina, the upper and larger end being held by the fingers and thumb of the left hand and the lower, or smaller end, in the same manner by the right hand. In Figure 88, the lateral arms of the pessary are being folded together by exerting pressure downwards by the forefingers and upwards by the thumbs and middle fingers. This is continued until the lateral arms meet. In Figure 89, a firm grip of the thumb and fingers of the right hand on the lower end of the pessary keeps the lateral arms together and thus the pessary has had its curve reversed. In Figure 90, the introduction is plainly shown. The reverse curve causes the upper end of the pessary to follow the posterior wall of the vagina to the posterior fornix. In Figure 91, the pessary has been released, has unfolded, and the upper end of the pessary has automatically taken its place posteriorly to the cervix.

It will be seen that this method obviates any necessity for re-adjustment after insertion. The pessary is easily removed by physician or patient by hooking one finger into the lower end. The pessary will fold itself as with-

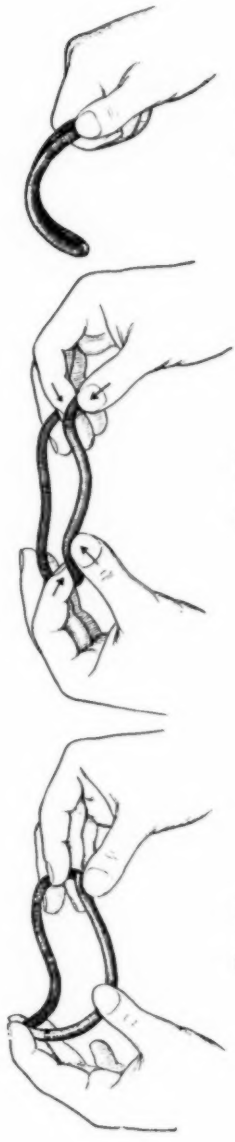

FIG. 87.

FIG. 88.

FIG. 89.

FIGS. 87, 88, AND 89. Method of bending pessary. (Findley, W. M. *Am. Jour. Surg.*, 33: 551, 1936.)

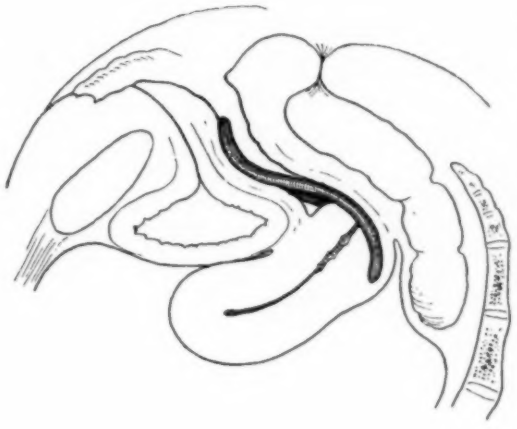

FIG. 91. Pessary in situ. (Findley, W. M. *Am. Jour. Surg.*, 33: 551, 1936.)

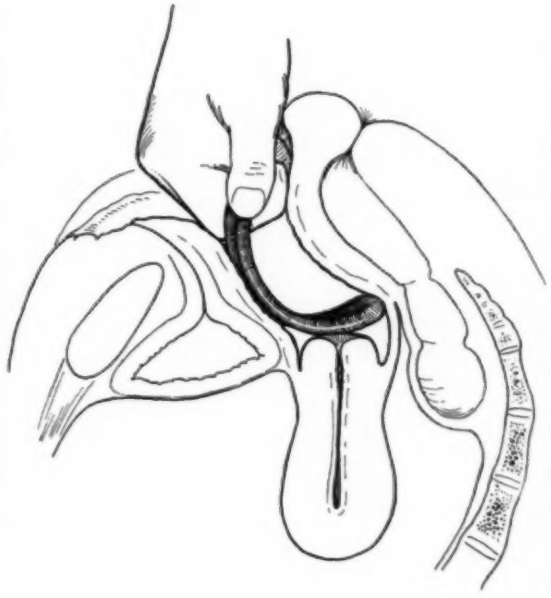

FIG. 90. Pessary inserted in vagina before release. (Findley, W. M. *Am. Jour. Surg.*, 33: 551, 1936.)

drawn. The discomfort and difficulties of application are thus largely eliminated. Various sizes may be tried to obtain proper fit.*

* FINDLEY, WM. M. Non-surgical treatment of retrodisplacement of the uterus. *Am. Jour., Surg.* 33: 546, 552, 1936.

Following the insertion of the pessary, the patient should return in one week for an examination. If she has had no discomfort and the uterus is well anterior the patient should be instructed to return every four to six weeks, preferably after a menstrual period. The pessary is then removed and cleaned, the vagina inspected for any pressure spots and the pessary reinserted.

A pessary as a rule should be worn for six months. Following the removal of the pessary, the patient is told to return in one week for a reexamination. If the uterus retains its normal position, she is to return in one month; if then it is still anterior, she is discharged. Following the removal of the pessary, the knee-chest position is continued night and morning and the patient is advised to sleep on her side or on her abdomen.

If on reexamination the uterus has again fallen back, it is replaced and the pessary is reinserted. If at the end of another six months, removal of the pessary again causes the uterus to fall back, producing symptoms, operation is advised.

When operative treatment is indicated, what procedures shall be used?

Examination of a standard operative gynecological textbook shows eighty pages devoted to a discussion of the various operations for retrodisplacement. The purpose of the operative treatment should be to restore the uterus to its normal physiological and anatomical conditions. The procedure which has given the best results at Harlem Hospital has been the Webster-Baldy operation combined with suspension of the ovaries and shortening of the uterosacral ligaments. In order to eliminate technical difficulties the uterosacral shortening is done before the Webster-Baldy operation.

Technique of Shortening the Uterosacral Ligaments. Any procedure for the shortening of the uterosacral ligaments must fulfill three demands:

1. It must keep the cervix well back in the hollow of the sacrum;

2. It must hold it there fairly permanently;

3. As the uterosacral shortening is only one of several operative procedures to be performed on the patient, it must not be time consuming.

FIG. 92. Placing of the sutures in shortening the uterosacral ligaments. Each is tied only after both have been placed. (Holden, F. C. *Am. Jour. Surg.*, 33: 553–557, 1936.)

Good anesthesia, the Trendelenburg position and an ample incision which extends down to the symphysis so as to secure good exposure, are absolutely essential for shortening the uterosacral ligaments.

Step 1. The intestines are walled off with the three pad technique.

Step 2. The uterus is pushed well forward, anteflexed, the

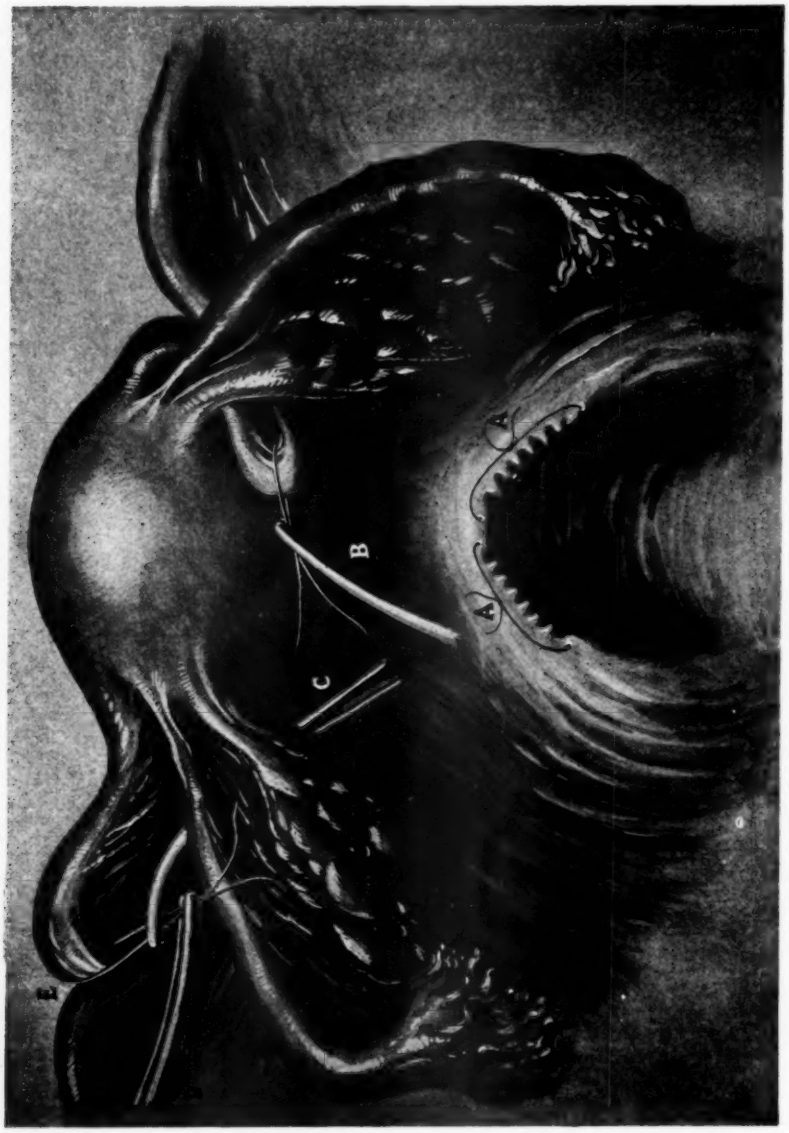

FIG. 93. The sutures shortening the uterosacral ligaments have been tied, A; a traction suture has been passed around the left ligament at E; a sharp pointed clamp has been introduced under the ovarian ligament and tube, C, and the traction suture, D, of the left round ligament grasped. The round ligament is then pulled through the opening made as is shown on the right side, B. (Holden, F. C. *Am. Jour. Surg.*, 33: 553–557, 1936.)

posterior wall of the uterus is grasped at the level of the internal os with a tenaculum and the cervix is drawn upward, making the uterosacral ligaments prominent. A continuous heavy silk suture on a curved Mayo needle is inserted from behind forward (Fig. 92) up to and including a bite in the posterior lip of the

cervix. This is inserted on both sides before being tied (Fig. 93A).* Both sutures are tied.

This procedure shortens the uterosacral ligaments from before backward thereby drawing the cervix well into the hollow of the sacrum. By using a nonabsorbable suture material, such as silk, the permanency of the shortening is assured.

The uterosacral shortening is then followed by the Webster-Baldy operation which seems to observe the basic anatomic as well as physiologic (intra-abdominal pressure) factors.

Step 1. A traction stitch (Fig. 93D) is passed under the round ligament at the junction of its middle and outer third and a clamp is applied to the suture.

Step 2. The ovarian ligament is grasped with an Allis clamp and a sharp pointed curved clamp is introduced under this ligament and under the tube (Fig. 93C) so as to emerge in the angle between the tube and the round ligament; here it grasps the traction stitch (Fig. 93E, C, D) and draws it backward thus pulling the round ligament under the tube and the ovarian ligament and through the opening (Fig. 93B).

Step 3. The same procedure is carried out on the other round ligament.

Step 4. The round ligaments are sutured to the posterior wall of the uterus with four or five interrupted fine silk sutures using a fine curved needle (Fig. 94B).

Step 5. Suspension of the ovaries. The ovarian ligament is grasped with an Allis clamp and drawn laterally as far as possible (Fig. 94D). The distal end of the round ligament (Fig. 94C) is grasped with an Allis clamp and drawn medially. A silk suture (Fig. 94E) is passed through the round ligament as far laterally as possible and through the ovary close to the origin of its suspensory ligament and tied (Fig. 94E and F). The same procedure is carried out on the other side.

Step 6. The abdomen is closed in the usual manner.

Failure following the Webster-Baldy operation may be due to one of several causes:

* HOLDEN, FRED. C. Surgical treatment of retrodisplacement of the uterus. *Am. Jour. Surg.* 33: 553–557, 1936.

1. *Retroflexion over the Round Ligaments.* If the round ligaments are sutured to the posterior aspect of the fundus at too low a level, the fundus may retroflex over these sutured

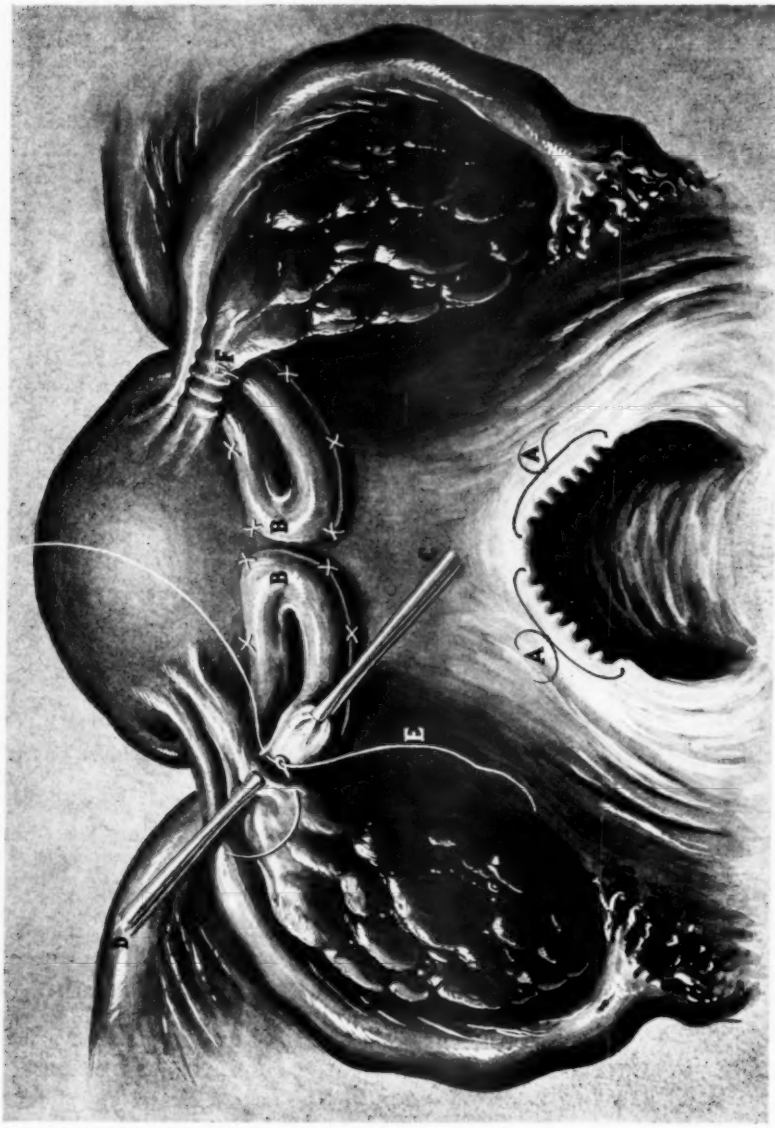

FIG. 94. The round ligaments have been sutured to the posterior aspect of the uterus at a level just below the insertion of the ovarian ligaments, BB. The ovarian ligament is grasped with an Allis clamp and drawn laterally, D. The distal end of the round ligament is grasped with an Allis clamp and drawn medially, C. A silk suture is passed through the round ligament as far laterally as possible and through the ovary close to the origin of its suspensory ligament, E, and tied, F. (Holden, F. C. *Am. Jour. Surg.*, 33: 553–557, 1936.)

round ligaments. The round ligaments should be sutured to the posterior wall of the uterus at a point which is on a level or just below the insertion of the uteroovarian ligaments. This is above the centre of the gravity of the fundus and will tend to hold the fundus forward.

2. *Recurrent Retrodisplacement.* This may be due to one of three causes:

A. *The Use of Absorbable Suture Material.* The catgut may absorb and no union take place between the round ligaments and the posterior wall of the uterus. This allows the ligaments to slip back to their former position with a recurrence of the retrodisplacement. Nonabsorbable suture material should be used throughout.

B. *Allowing the Cervix to Remain Forward.* Some of the retrodisplacements recur because the cervix is allowed to remain anteriorly after the fundus has been brought forward by the Webster-Baldy operation. Shortening of the uterosacral ligaments according to the technique described will hold the cervix well back in the hollow of the sacrum. This is an important additional factor in securing satisfactory results. In studying the failures in a series of operations, it was observed that recurrences were found in those patients who on discharge did not have the cervix well back in good anatomical position.

C. *Non-repair of Relaxed Vagina.* Laceration of the pelvic diaphragm and relaxation of the vaginal outlet will allow the uterus to fall backward again after operation as well as after removal of a pessary. In order to restore proper anatomical relationship it is of the utmost importance that the anterior and posterior vaginal walls, if relaxed, should be repaired. This will help maintain the uterus in its anterior position.

3. *Numerous painful adhesions between intestines, sigmoid, omentum and posterior wall of the uterus* are sometimes found. These can be prevented by causing a minimum of trauma to the tissues. The round ligaments are held by catgut "guy ropes" (Fig. 93B and E) and they should not be crushed with a clamp. The round ligaments are sutured to the posterior wall of the uterus using fine curved needles (Ferguson needles size 2, round point, half curved) and fine silk.

4. *Postoperative Pain Due to Tender Prolapsed Ovaries or to Ovaries Which Have Become Adherent to Each Other in the Midline.* The Webster-Baldy operation is the only operation for retroversion which at the same time elevates the ovaries

and tubes. One finds only too often that it is the prolapsed ovary which has been giving the symptoms rather than the retroversion. Simple elevation of the uterus in certain cases will not suspend the ovaries owing to long uteroovarian ligaments. This is corrected by drawing the round ligaments backward just below the uteroovarian ligament (Fig. 93B and C). Although the ovaries will thereby be drawn upward, it will not prevent them from coming in contact with each other. To prevent this, the distal end of each round ligament of the uterus is grasped with an Allis clamp and drawn laterally (Fig. 94C and D). When the round ligament has been drawn as far medially as possible, a silk suture is passed through the round ligament and through the ovary close to the origin of its suspensory ligament (Fig. 94E). In this way the round ligaments exert a constant pull on the ovaries and keep them well suspended and separated from each other (Fig. 94E and F).

Axiom: One learns not by one's successes, but by a critical and impartial analysis of failures. Careful attention to each detail is frequently the factor which will make the difference between failure or success.

Question: Should the pessary be boiled for sterilization?

Answer: No; a boiled pessary is always round; boiling a hard rubber pessary should only be done if you wish to change its shape.

Question: How can you make slight changes in the shape of the pessary?

Answer: Cover the pessary with vaseline and heat the part to be molded over an alcohol flame until it is mallable, then bend the part to the desired shape. The vaseline keeps the hard rubber from burning.

Question: What is the effect of pregnancy on the Webster-Baldy operation?

Answer: When the operation has been performed as described there should be no recurrence of the retrodisplacement even after pregnancy.

Question: What should be done if the pessary causes a pressure sore in the vagina?

Answer: Heat the pessary as described and bend the particular spot in such a way as to remove the undue pressure. The red spot in the vagina should be "touched up" with 10 per cent silver nitrate or pure pyroligneous acid.

Question: How is a pessary cleaned?

Answer: It is placed in a basin of cold lysol solution and scrubbed with a brush following which it is washed in cold water.

Chapter VII

MENSTRUATION AND ITS DISORDERS

A study of the histories of any large series of gynecological cases would show that deviation from the normal menstrual cycle is one of the most frequent complaints presented for treatment. The endometrium is the background of normal and abnormal menstruation. In order to understand the various types of bleeding properly it is absolutely essential to have a clear conception of (1) the normal histological changes that the endometrium undergoes and (2) the glandular interrelationship which is responsible for these changes.

The normal endometrium undergoes a regular cycle of histological changes every twenty-eight days. This cycle may be represented by the circumference of a circle which is divided into 28 equal parts representing the days. To study any regularly repeating cycle it is necessary to begin at some fixed point. The first day of menstruation is the only fixed point in the entire menstrual cycle. A woman always knows when she begins to menstruate; she may not know the exact time when she stops, but she always does know the first moment she starts. Although the first day of menstruation is used as the fixed point, the beginning, it must be recognized as a fact, that menstruation proper is the end of the normal physiological cycle and not its beginning.

In the normal 28 days cycle a woman menstruates for four days. The six-day period just prior to menstruation is called premenstrual, the six days just after menstruation, postmenstrual and the twelve days in between, the interval period (Fig. 95). This division is purely arbitrary and is used as an aid for study.

There are two common misconceptions with regard to the endometrium that must be corrected:

The endometrium is not a true mucous membrane as it has no submucosa. If it had a submucosa, with every curettage

and with every delivery the entire endometrium would be lost. Nothing would be left from which the new endometrium could regenerate.

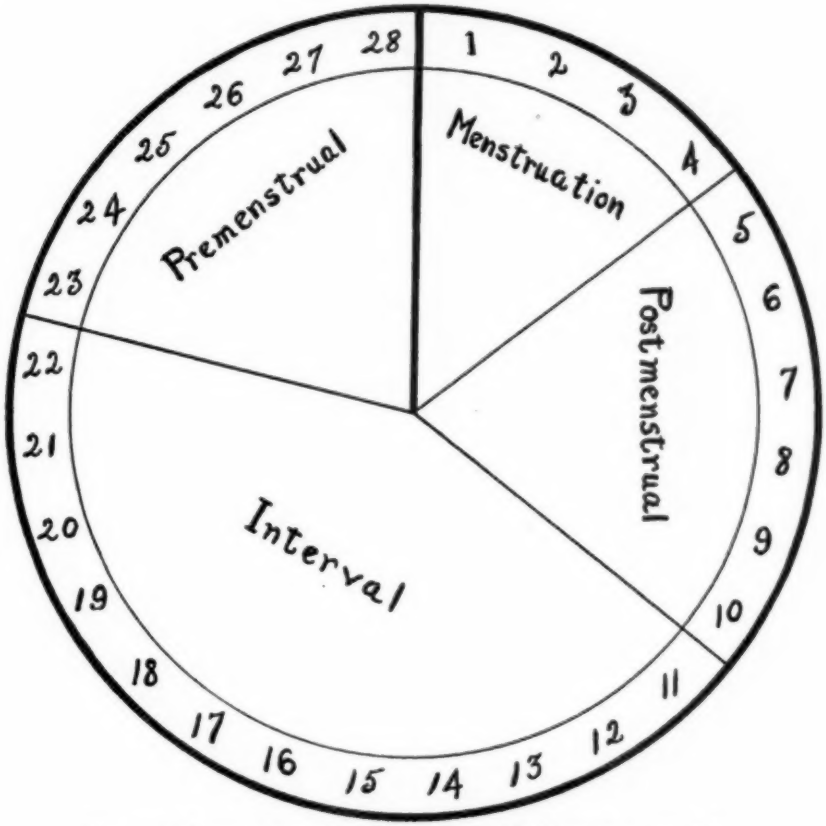

FIG. 95. Menstrual cycle arbitrarily divided into four periods.

The second misconception is that there is a resting stage or period in the endometrial cycle. This is not true. There is no resting period. The endometrium is an ever-changing structure. It may not change in its entirety every day, but there is some change in some part of the endometrium daily.

PREMENSTRUAL ENDOMETRIUM. This is the most complex form the endometrium assumes. Grossly, it is composed of glands and stroma. The glands can be divided roughly from their general appearance into three parts: the mouth of the gland (Fig. 96-1), the tortuous part of the gland, (Fig. 96-2), and the inactive fundus (Fig. 96-3).

MOUTH OF THE GLAND or compact layer of the endometrium. The epithelium lining the cavity of the uterus is a ciliated high columnar epithelium having a central nucleus and a granu-

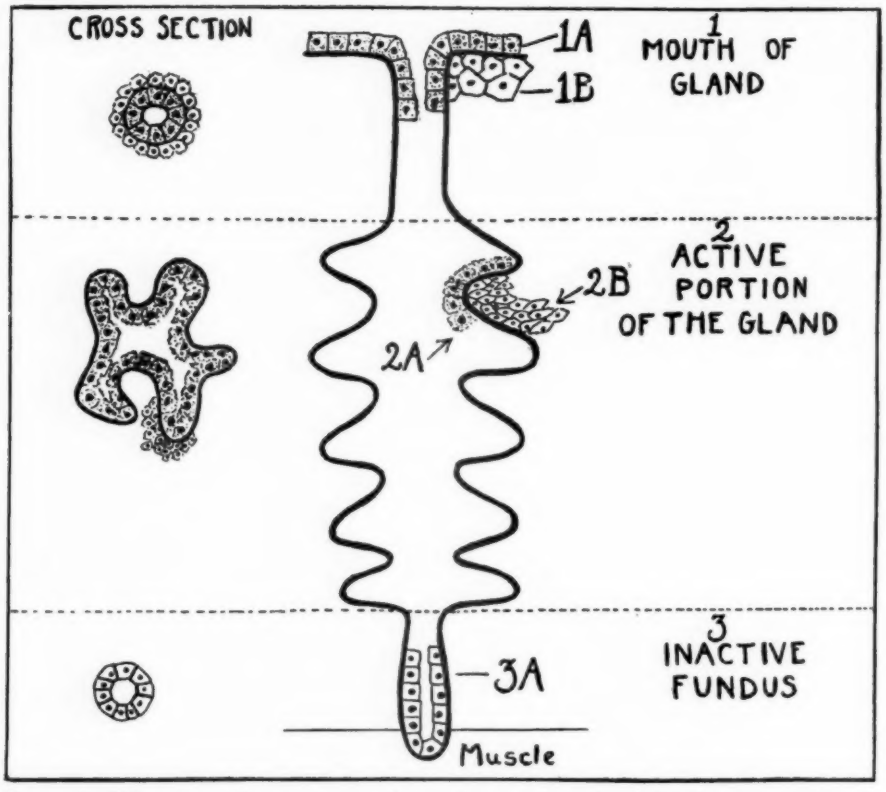

FIG. 96. Diagrammatic representation of a uterine gland in the premenstrual secretory phase. 1A, epithelium lining cavity of the uterus and mouth of the gland; 1B, edematous connective tissue, stroma cells of the compact layer; 2A, hyperplastic epithelium of the tortuous part of the gland; 2B, spindle cell shape of stroma in this region; 3A, low columnar epithelium of the inactive fundus.

lar cytoplasm (Fig. 96-1-A). The mouths of the glands are also lined by a high columnar epithelium with a central nucleus and glandular cytoplasm. Underneath the surface epithelium and between the mouths of the glands are found larger polyhedral connective tissue cells with central nuclei and clear cytoplasm (Fig. 96-1-B). These large connective tissue cells become the decidual cells of the pregnancy endometrium. It is this layer which forms the compact layer of the pregnancy endometrium; it is in this layer that the ovum imbeds itself in the earliest stages of pregnancy. There is very little or no

intercellular structure found in this layer except just before menstruation when there is usually a marked infiltration with lymphocytes, polymorphonuclear leucocytes and red blood cells. There are few blood vessels in this area. If any blood vessels are found, they are terminal endothelial lined spaces.

SPONGY LAYER OF THE ENDOMETRIUM, or tortuous part of the gland (Fig. 96-2). This comprises the active portion of the gland. The gland in this area assumes a corkscrew appearance. This is due to an attempt on the part of the gland to accommodate itself to the increased space required for the hypertrophied lining epithelium. The lining epithelium is high columnar with a central nucleus and a granular cytoplasm. It has the appearance of a stimulated epithelium. The epithelial cells look as if they were ready to burst into the lumen of the gland. They are not sharply demarcated from the lumen of the gland (Fig. 96-2A). In the lumen of the gland there is some secretion, which has been found to contain trypsin. This is a proteolytic enzyme which becomes activated in an alkaline medium. The cavity of the uterus is normally slightly alkaline. When this trypsin is released into the cavity of the uterus, it becomes activated and proteolytic digestion takes place.

The stroma (Fig. 96-2B) of this layer, is not so markedly hypertrophied as is the stroma of the compact layer. The connective tissue cells have a central nucleus and have retained their oval or spindle cell shape. There is a moderate amount of plasma (edema) between these cells. With the approach of menstruation one finds white blood cells, red blood cells and blood in the interstices. The blood vessels in this area are more numerous than in the compact layer. They are the terminal capillaries; they may be simple endothelial lined spaces or may have the beginning of a musculature in the walls.

INACTIVE FUNDUS OR FUNDAL LAYER (Fig. 96-3). This presents itself as a perfectly straight tubular gland lined by a low columnar epithelium (Fig. 96-3A) taking a very deep stain with hematoxyline. *The younger the cell, the more hematoxylin it takes.* They are the youngest epithelial cells of the entire gland. The stroma of this layer is a very dense connective

tissue; the arterioles are terminal arterioles with definite muscular walls. The gland fundii dip down into and rest upon the musculature of the uterine wall. *There is no submucosa.* When pregnancy takes place the inactive straight fundal glands disappear and become dilated.

Diagnostic Point: When a patient presents herself with an amenorrhea and examination of the cast-off or curetted endometrium shows dilated fundal glands and no trophoblasts, a diagnosis of tubal pregnancy must be considered.

As a result of certain endocrinological changes menstruation occurs. If a cup were placed in the vagina of a menstruating woman and all the material discharged from the cervix collected and examined chemically and microscopically, one would find on the *first day* that the first discharge would be a moderate amount of serum. Many women complain that just before the bleeding actually starts, they feel quite wet. This is apparently due to a discharge of serum from the hypertrophied connective tissue cells of the compact layer. This serum would be followed by a considerable amount of blood, some tissue, and an appreciable amount of trypsin. On the first day, uterine epithelium was demonstrated in the centrifuged specimen, on section in 50 per cent of 100 cases studied by Geist.

On the *second day* there is a definite increase in the amount of blood and in the amount of uterine stroma and epithelium discharged. There is a definite diminution in the amount of trypsin. This is due to the fact that trypsin is not being produced during menstruation. Trypsin is produced by the glands from one menstrual period to the next.

On the *third day* there is a moderate amount of blood, some uterine stroma and practically no trypsin.

On the *fourth day* practically nothing but blood is found.

If a daily examination of the endometrium could be made during menstruation, one would find that during the first day or so the upper part of the gland (the compact layer) with the accompanying edematous connective tissue, is discharged. On the second day part of the active part of the gland accompanied by its connective tissue stroma, is discharged. On the third and

fourth days there is a gradual diminution in the cellular elements thrown off until nothing but blood is discharged. So that with the end of menstruation and the beginning of the postmenstrual period all that is left of the premenstrual gland is the inactive fundus.

Does this loss of tissue happen with every menstruation? No.

Does this change take place in all the glands with each menstruation? No.

The loss of endometrium may be as complete as described throughout the entire uterine cavity or it may go to the opposite extreme with practically little or no loss or only a patchy loss of the endometrium. In some areas the epithelium may simply lose its hypertrophied appearance, the glands straightening out and the connective tissue loses its edema.

Postmenstrual Endometrium. The endometrium has shrunk very much in size and the cells have lost their marked appearance of stimulation. Many areas of denudation are present throughout the endometrium. As soon as the destructive process ceases, epithelial proliferation begins to cover the raw areas. This starts from surviving islets of epithelium and from the broken ends of the fundal glands. This proliferation of the remaining epithelium sets in very rapidly so that at about the end of the postmenstrual period the inactive fundii have regenerated, forming a simple tubular gland, and the epithelium has spread over the surface so that the uterus is again lined by epithelium on or about the tenth day after the first day of menstruation.

Interval Endometrium. With the beginning of the interval period the glands begin to show definite growth. They become elongated and the epithelium gradually becomes hypertrophied so that by the end of the interval period, there is a definite division of the gland into three parts: the mouth, the active portion, and the inactive fundus. Toward the end of the interval period secretion appears in the gland. The transition from the interval stage to the premenstrual one is very gradual and one frequently will find great difficulty differentiating a

late interval endometrium from an early premenstrual one.

What causes these changes?

What is the endocrinological background of all this?

There is no doubt that something in the ovary controls the various changes in the endometrium. It has been definitely established that if both ovaries are removed, menstruation ceases. But what particular structure in the ovary controls the endometrium?

The primordial follicle, containing the ovum and the various structures arising from it seem to control the changes in the endometrium. With the beginning of the cycle several of these primordial follicles show signs of activity (Fig. 97). As the primordial follicles proliferate they give off a secretion which is called female sex hormone (Frank), folliculin, theelin, etc. In this discussion the product will be called theelin. Somewhere between the fifth and the seventh day after the first day of menstruation, one of these primordial follicles begins to show signs of marked proliferation and goes on to the formation of a Graafian follicle. With each menstrual cycle, as a rule, only one Graafian follicle is formed.

As the Graafian follicle ripens and extrudes the ovum, the corpus hemorrhagicum and later the corpus luteum form. With the formation of these structures the other primordial follicles which had begun to proliferate, degenerate and disappear and the production of theelin is assumed by the corpus luteum (Fig. 97).

The ovum is extruded about the twelfth day after the beginning of menstruation. With the expulsion of the ovum, the Graafian follicle collapses, hemorrhage takes place into it with the formation of the corpus hemorrhagicum. Large polyhedral cells with central nucleii and granular cytoplasm, containing a lipoid known as lutein, grow into the hemorrhagic area and by the twenty-second day the corpus luteum of menstruation is fully formed. The ovum during this time has continued its passage down the tube and if it has not become impregnated it dies on or about the twenty-sixth day. The

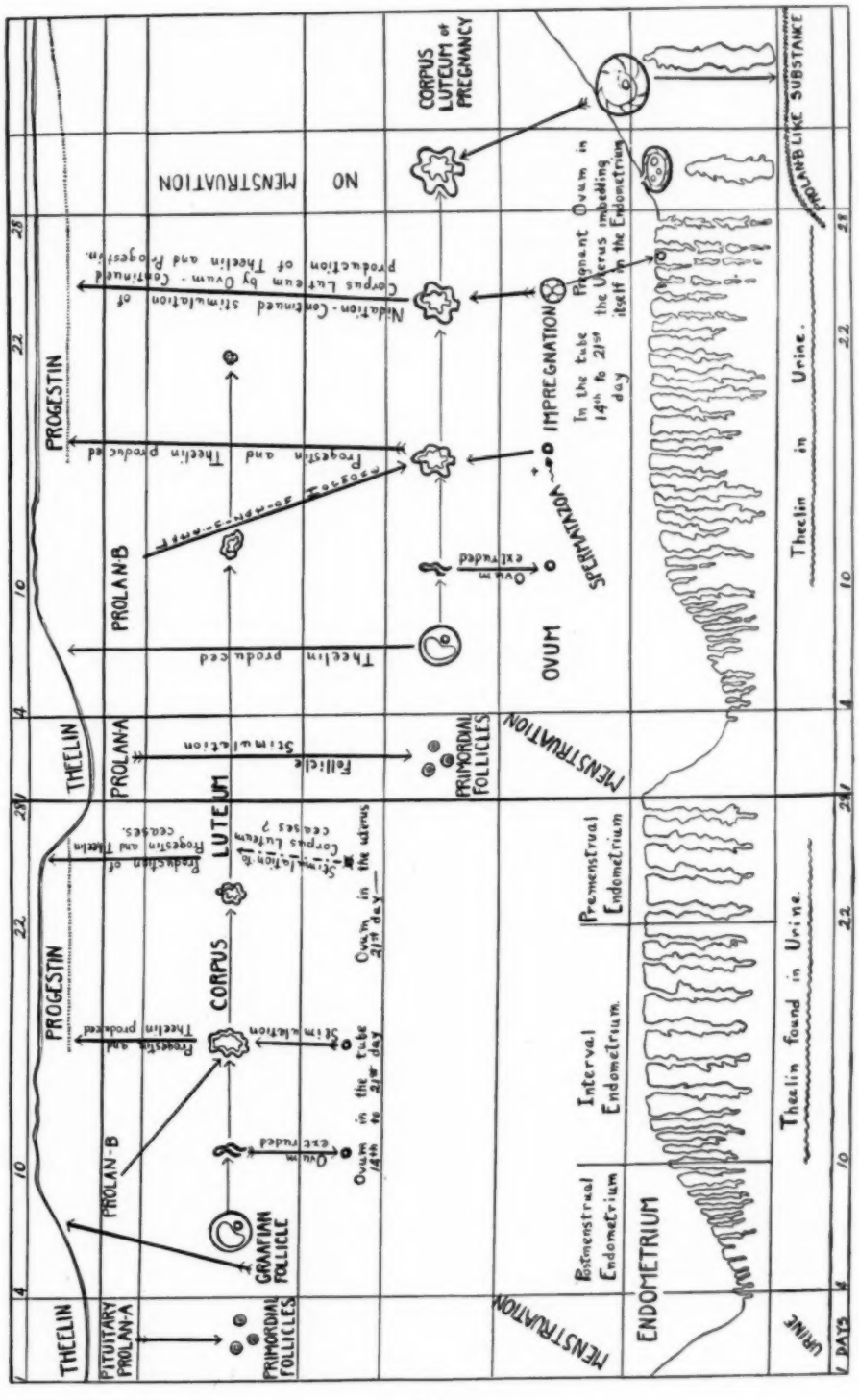

FIG. 97. Schematic representation of the endocrinological background of menstruation. (*Modified from Dickinson.*)

lutein cells besides producing theelin, gives off another secretion known as progestin (Fig. 97). The corpus luteum is supposed to function (if the ovum does not become impregnated) until about the twenty-sixth or twenty-seventh day after the first day of menstruation, then regression takes place.

What correlation is there between these ovarian changes, the endocrinological changes and the changes in the endometrium?

With the end of menstruation there is practically no theelin in the blood. The early postmenstrual changes can be ascribed to the normal healing or regenerative processes. With the proliferation of the primordial follicles and the formation of the Graafian follicle, the theelin content of the blood gradually increases until it reaches a plateau. Theelin causes the late postmenstrual changes and all other changes in the endometrium up to the end of the interval period. Theelin also sensitizes the endometrium to the action of progestin. Progestin produces the premenstrual changes, the secretory phase, in an already sensitized endometrium. (Theelin and progestin are both produced by the corpus luteum.)

What causes these various changes to take place in the ovary?

The stimulus which initiates follicular activity comes from the anterior pituitary lobe and is called prolan A (Fig. 97). The hormone which causes the luteinization of the follicular epithelium also comes from the pituitary gland, is called prolan B. The ovum apparently maintains the life of the corpus luteum. With the death of the ovum, stimulation to the corpus luteum ceases; with its cessation, the production of progestin and theelin ceases, and the stimulation to the endometrium stops. With the cessation of the stimulation, the edema of the compact layer of the endometrium is discharged and the mouths of the glands open; trypsin is expelled into the cavity of the uterus and proteolytic digestion takes place. The blood vessels rupture and menstruation occurs.

The corpus luteum ceases to function with the death of its particular ovum. If pregnancy takes place, that ovum imbeds

itself into the endometrium and continues to stimulate its corpus luteum forming the corpus luteum of pregnancy (Fig. 97). The corpus luteum in turn continues to stimulate the endometrium for two to three months. If the corpus luteum of pregnancy is removed in less than three months, the patient may abort. The placenta when formed assumes some of the functions of the corpus luteum.

Menstruation without ovulation can take place, in such cases the endometrium does not develop beyond the late interval stage, e.g., the twenty-second day endometrium. This is due to the absence of progestin. A premenstrual hyperplasia (secretory phase) will not be found if a patient who menstruates without ovulating, is curetted.

Bleeding around the middle of the period might be due to fluctuations in the folliculin content of the blood, as the rupture of the Graafian follicle takes place at this time.

The theories as to the cause of menstruation are many, but the true factor is still a matter of much speculation.

Theory 1. There is a gradually increasing amount of evidence tending to prove that menstruation is due to a change in the theelin content of the blood, a lowering of the theelin concentration level in the blood (Fig. 97). Just what this level is, how much theelin shall be called the normal amount, is not definitely known.

What is the presumed endocrinological mechanism which brings about this theelin level?

The primary stimulation factor comes from the pituitary gland, prolan A, which is a follicle stimulating hormone, causing several primordial follicles to proliferate. One of these follicles for some unknown reason goes on to form the ripe follicle, the Graafian follicle; the other degenerate and disappear.

The summation of the internal secretion of these follicles produces a sufficient amount of theelin to inhibit this activity of the pituitary gland and to stop further follicle development. With the rupture of the Graafian follicle, the second pituitary hormone is secreted, prolan B, which is the luteinization hor-

mone and produces the luteinic changes in the follicular cells of the Graafian follicle. The lutein cells produce theelin and thus maintain the necessary level. About one to two days before menstruation begins the corpus luteum begins to retrogress, the theelin level is lowered and menstruation results. With the lowering of the theelin level the inhibition on the pituitary is removed and prolan A again functions and the cycle is repeated.

Theory 2. Ovulation and menstruation.

For many years menstruation was regarded as the abortion of a nonimpregnated ovum—no ovulation, no corpus luteum —no corpus luteum, no menstruation—was Robert Meyer's dictum. The summation of the internal secretion of the primordial follicles stimulates the endometrium until the Graafian follicle ruptures and the ovum is extruded. The corpus luteum is then formed. As long as the ovum remains alive it stimulates the corpus luteum which produces theelin and progestin. With the death of the ovum, retrogression of the corpus luteum occurs. The production of theelin and progestin ceases, and the stimulation which produced the thick, hyperplastic premenstrual endometrium stops. The decidual-like cells (subepithelial connective tissue cells of the stroma) which became hypertropied as a result of the stimulation by progestin kept the mouths of the glands closed. With the stoppage of this stimulation the connective tissue cells lost their edema. Some women state that a day or so before they actually begin to menstruate, they feel quite moist as a result of the loss of this fluid. The loss of edema causes the mouths of the glands to open, the contained trypsin is discharged into the uterine cavity and proteolytic digestion begins. Trypsin also prevents the coagulation of the blood. With the stoppage of the stimulation and the release of pressure on the outside of the vessels, the thin walled structures rupture, bleeding takes place—this plus the trypsin digestion produces menstruation.

How long will the ovum live after being discharged from the Graafian follicle?

It is probably viable and vulnerable to impregnation for only a short period, but its effect on the corpus luteum, if the ovum does not become impregnated, lasts about fourteen days.

A patient presents herself with a menstrual disorder. How shall one go about establishing the proper diagnosis?

Before a menstrual disorder can be attributed to any functional disturbance, all other organic or pathological conditions which might cause that disorder must be eliminated. The history should be taken with special reference to the periodicity of the symptoms so as to fix them in relationship definitely with, or as a cause of, the menstrual disturbance. The physical examination should rule out any gross pathological condition. Following this certain tests can be performed in an attempt to fix the responsibility for the menstrual disorder.

Tests and Examinations

1. *The daily theelin variations* in the blood and urine of the patient from one menstrual period to the next, can be estimated. This is very expensive, costing about $150. for one month's tests. The information obtained, however, may be very valuable. It is by far the most accurate method of determining exactly how much ovarian function is going on. Clinically one may, by inference, as a result of a careful history and physical examination, judge hypoovarian function by searching for signs of juvenilism (see Chapter VIII, Sterility).

2. *Basal Metabolism.* The close relationship between the thyroid and ovary requires a complete knowledge of the state of function of that gland. Hypofunction or hyperfunction of the thyroid may cause ovarian dysfunction.

3. *Examination of the Pituitary.* There is no accurate way of determining the function of the pituitary gland. One must work by inference depending upon tests and examinations to give a lead to the pituitary function. *X*-ray examination of the sella turcica is done: What is its size? Is it too small? Is it too large? Is any tumor demonstrable? A tumor may mean hyperfunction or pressure atrophy with loss of function. Are the

fundi normal? Has there been encroachment upon the visual fields? What is the sugar tolerance of the patient? All of these examinations and tests give indirect information as to pituitary function.

4. *Curettage.* This is a simple and yet a very valuable examination. It is practically an office procedure.

These tests are time-consuming, expensive and after all the tests are performed, the question as to whether an accurate endocrinological diagnosis has been established is frequently in doubt.

If examination of the curettings removed two or three days before the expected menstrual period shows the presence of a premenstrual endometrium it is indicative of a previous ovulation and the presence of an ovum. A premenstrual (secretory phase) endomentrium shows the presence of progestin; the latter is produced by a corpus luteum which structure arises from a Graafian follicle following the extrusion of an ovum. From the absence of a secretory endometrium one must infer that ovulation has not occurred. As all chemical tests for progestin have failed, to date, curettage is the only means of demonstrating the occurrence of ovulation.

5. *Allergy.* Recently many menstrual disorders as well as other gynecological conditions have been ascribed to allergic phenomena. The diagnosis of these allergic phenomena is as yet very difficult. One must, for the present, until further data is available, depend upon the history and periodicity of the attacks as a lead to this diagnosis.

Axiom: You cannot make a diagnosis unless you know the disease and think of it.

In those cases where a fairly accurate endocrinological diagnosis can be established, rational organotherapy is very problematical. It is expensive and frequently ineffectual. It is difficult to maintain proper standards with the commercial endocrine substances used because so many of them deteriorate rapidly and very often the dosage used is much less than the one marked on the container. Wherever possible, synthetic, stable products should be used. Unfortunately there are as yet

no such products for the various ovarian hormones. One of the few organotherapeutic procedures that has proved to be of uniform value has been the use of thyroid gland substance in cases with low basal metabolic rates. There is no question that the basal metabolism can be raised with thyroid substance.

Amenorrhea

It is of paramount importance in all menstrual irregularities in young women, to impress the patients and their mothers with the fact that there is nothing in the body that must be expelled every month with menstruation and that the condition is compatible with good health and does not necessarily mean some serious constitutional ailment.

Clinically the amenorrheas may be divided into two large groups:

1. Primary amenorrhea, those patients who have never menstruated, usually young women or adolescents;

2. Secondary amenorrhea, those who have menstruated regularly or irregularly for a period of time and now do not menstruate.

In the primary amenorrheas one must first exclude all abnormalities of the gynecological tract such as absence of vagina or uterus, atresia of the vagina, stenosis of the cervix, etc.

What can be done for these patients with primary or secondary amenorrhea? A large percentage of these patients will ultimately regulate their own internal secretory functions. It is only necessary to tide them over this period psychologically until the glands begin to function normally. Good hygiene, outdoor exercise, "put them out to grass" (Cragin), stop most indoor work, diminish all social activities, create regular bowel and sleeping habits, all of these will help the patient establish her normal physiological-endocrinological balance.

It is in this type of case that it is essential to know if the ovaries are functioning. Is any theelin being produced? Frank divides these cases into three groups:

A. *Subthreshold Group.* Those patients showing a regular curve of the accumulation of theelin (female sex hormone) in the blood but not sufficient to produce the changes necessary to cause menstruation;

B. *Negative Blood Cycle* (high theelin content in the urine). Those cases showing no accumulation of hormone in the blood on repeated examinations and yet show an excess in the urine (low renal hormone threshold).

C. *Negative Blood and Urine Cycle.* Those showing no demonstrable quantity of theelin in either their blood or urine. Clinically B and C can be considered together.

What can be done to produce and maintain the proper theelin concentration in the blood of a patient?

The maintenance of a proper theelin level is difficult and uncertain because the normal level in any patient is not known and therefore the amount of theelin necessary to maintain this level cannot be known. All examinations and tests for theelin are made on the basis of rat units, that is to say, the quantity of theelin necessary to produce an estrus cycle in the vagina of an immature rat. The dosage that is required to produce the same sort of changes in the human is very problematical. If the dosage is gauged on the relative weights of the rat and the human, one would have to use 1000 times the amount used on a rat. In other words, to maintain the supposed proper female sex hormone level in the blood of a human would require 200,000 rat units equally spread over a one month period. This is very expensive and the results are very doubtful.

In the subthreshold group theelin may be given over short periods of time; 10,000 rat units three times a week for two weeks and if no bleeding occurs it can be repeated in two weeks. This may be sufficient to produce a stimulation of the endometrium and cause bleeding (not regular menstruation). This bleeding has an excellent psychological effect on the patient and helps tide the patient over until regular function is reestablished. This however, should not be continued for too long a period of time, nor be too excessive, as it may cause destructive changes in the ovaries.

What can be done for those patients showing no theelin in the blood? If large doses of theelin are given patients with non-functioning ovaries, an interval endometrium can be produced with bleeding. There is no better way of explaining this effect of large quantities of theelin in the non-menstruating woman than by quoting Novak who says, "It is like pushing the pendulum of a run-down menstrual clock for a single, idle, incomplete beat, when what we need is something to wind the clock."

Has anything actually been accomplished therapeutically by giving large doses of theelin? An interval endometrium has been produced, bleeding has occurred, but the ovaries have not functioned in any way. To have this patient bleed again it will be necessary to give her another course of theelin so as to produce another endometrium. Therapeutically, what is wanted, is not something to cause a single bleeding from the endometrium but something which would stimulate the ovaries to function, e.g., to "wind up the ovarian clock" and have it continue to function.

The dominant role of the anterior lobe of the pituitary over ovarian function is generally accepted. This would suggest that the key to "winding up" the menstrual clock might be sought in the anterior hypophysis.

The use of large doses of Prolan A, which is the follicle stimulating hormone, would be the ideal approach. However, prolan A as yet has not been produced commercially, nor has it been definitely isolated.

The usual pituitary product sold is a prolan B-like substance obtained from the urine of gravid women. This contains a product similar to the luteinizing principal of the pituitary gland. This prolan B-like substance acts on the follicular epithelium of the ovary which is supposed to become luteinized and produce progestin. This in turn can act only on an endometrium sensitized to it by large doses of theelin. This product would therefore be of no value in amenorrhea, particularly when due to non-functioning ovaries.

What can be done to "wind up" the menstrual clock?

Anything which would start the ovaries functioning "winds up the ovarian clock" and cures the amenorrhea. *Very small doses of x-ray stimulation to the pituitary and ovaries may start them functioning, and thus "wind up the menstrual clock" and cure the amenorrhea.*

The treatment of the secondary amenorrhea, oligomenorrhea or hypomenorrhea depends to a large extent upon the desire of the patient to have children. If there is no desire for pregnancy, no treatment other than psychotherapy need be instituted, as these conditions are compatible with a normal life. Should pregnancy be desired, the regular daily estimation of the theelin content of the blood and urine becomes of tremendous importance. These patients as a rule, belong to the "subthreshold" group of Frank. Their ovaries are functioning but are not producing enough theelin to cause the normal menstrual cycle. These patients can be helped to a fairly normal amount of bleeding (empirical) by giving 10,000 rat units of theelin every day or every other day for two weeks before the expected date of menstruation. This may cause a more profuse bleeding in the oligomenorrhea and hypomenorrhea patients. This procedure may be carried out every month for six months, in the hopes that the patient may reestablish her own interglandular relationship and thereby raise the theelin content of her blood spontaneously. Its effect is mainly psychological during the period of readjustment. As a last resort one may use *very small* doses of *x*-rays to stimulate the ovaries to function normally.

Menorrhagia, Metrorrhagia

For therapeutic purposes these patients should be divided into their respective age groups, adolescent, middle age and menopausal. Clinically they may be grouped according to their symptomatology into the moderate or severe bleeding, depending upon the amount and frequency of the bleeding and the degree of anemia present.

The irregular bleeding of the adolescent is usually due to some endocrinological imbalance. These patients should be

psychologically assisted over this period. The prognosis is good as most of these cases correct themselves spontaneously.

The profuse irregular bleeding of the adolescent may be caused by a hyperproduction of theelin with a non-ovulatory endometrium, e.g., bleeding from an endometrium which has not entered the premenstrual or secretory phase due to the absence of one of the ovarian hormones, progestin. Progestin is one of the hormones produced by the membrana granulosa cells of the Graafian follicle after ovulation has taken place and luteinization has begun. In such cases good results should be obtained by the use of prolan B, the luteinization hormone of the pituitary or progestin itself, but as these products cannot be obtained commercially* a prolan B-like substance obtained from the pregnancy urine is used. This substance is supposed to cause a luteinization of the follicular epithelium in the follicle. This has been tried but not proved on human ovaries. This luteinized epithelium produces progestin which in turn produces the premenstrual or secretory endometrium. Theoretically this should correct the irregularity. Therapeutically (empirically) the patient should receive 200 to 400 rat units of the prolan B-like substance every day for five days before the bleeding starts. If the patient has menorrhagia, the injections may be given with the onset of menstruation. If the patient is irregular, the injections may be begun at any time. The injections of the prolan B-like substance may have to be repeated several times before the flow regulates itself.

In cases of severe hemorrhage in adolescence, curettage may be performed. This will usually help the condition for a short time. During this period endocrine therapy should be tried. If the bleeding recurs and is again severe, a temporary sterilization dose of *x*-rays should be tried; with the hope that when menstruation returns the interglandular relationship will have been reestablished and the periods will be normal. One must watch these cases very carefully and not allow them to get out

* Some of the commercial houses are selling a product which they claim contains the prolan B of the pituitary gland. Others claim they have been able to isolate progestin for commercial use. Both products are still in the experimental stage clinically.

of hand. The bleeding, when excessive can cause such severe anemia as to require repeated transfusions.

Goldberger and Peck* have reported good results in these bleeding cases with the use of snake venom. Their "Method of Administration" is described verbatim.

> The moccasin venom was used in a 1:3000 dilution with sterile sodium chloride (normal) containing 1:10,000 merthiolate.
>
> All injections were given subcutaneously. The initial injection was 0.5 c.c. and subsequent injections were rapidly increased to 1 c.c. (by the third injection). The interval between administrations of the venom depended on the severity of the bleeding. It was advisable to give as much venom as possible the first ten days, because at about that time the majority of patients developed a sensitivity to the venom which necessitated a decrease in the amount until desensitization was accomplished.
>
> There was a distinct quantitative relationship between the desired clinical effect and the amount of venom given. In cases with marked bleeding, 1 c.c. of 1:3000 dilution was given daily or even twice a day until the hemorrhage was controlled. In a number of the patients, daily injections were given at the beginning of the treatment. After the bleeding had been controlled the interval between injections of venom was increased so that only two or three treatments were given weekly.
>
> If the subsequent menstrual period approximated the normal, two injections a week were administered for at least three normal menstrual periods. During the course of the treatment a maintenance dose had to be established.
>
> In a number of the treated individuals a period of six months to one year of normal menstruation occurred after the venom therapy had been discontinued. When metrorrhagia or menorrhagia recurred and venom was again administered, the initial dose was 1 c.c. If such a patient reported for treatment early enough, several 1 c.c. injections given two or three times weekly were found to be sufficient to bring about normal menstrual bleeding. It has also been noted that the course of injections necessary to control recurrences of bleeding was shorter than that necessary at the beginning of the treatment.

When patients in the middle age or menopausal group present themselves with moderate to severe bleeding, curettage as a therapeutic and diagnostic precedure should be tried early. The most important single therapeutic agent in the treatment

*Goldberger, M. A. and Peck, S. M. Additional data on treatment of uterine bleedin with snake venom. *Am. Jour. Gynec. and Obst.*, 33: 469–472 (March) 1937.

of functional uterine bleeding is the curette. Curettage usually stops the excessive bleeding for several periods and also allows a careful examination of the tissue obtained to rule out the possibility of an undiagnosed incomplete abortion or an early carcinoma of the fundus. One may also in this way diagnose a non-ovulatory hypertheelin bleeding with a late interval endometrium, e.g., "Swiss cheese endometrium." The middle age group with excessive bleeding who are desirous of retaining their procreational capacity are treated the same as the adolescent with moderate to severe bleeding. If however, no further pregnancies are desired these patients should be treated like the menopausal group.

In the menopausal group, a castration dose of *x*-rays is frequently given without trying any of the mentioned treatments other than curettage. Radium has the same effect as *x*-rays but has the added effect of destroying the endometrium. Surgery in the form of oophorectomy or hysterectomy can be practised as a last resort.

Dysmenorrhea

No attempt will be made to enumerate the various etiological factors or theories of dysmenorrhea. The problem will be approached from a purely clinical standpoint.

A patient presents herself complaining of dysmenorrhea. It is presumed that all gross pathological lesions have been ruled out. The severity of her complaint may be judged by the amount of incapacitation it causes. Is she able to carry on her normal work or does she have to stay in bed with each period?

What shall be done for her?

A fair number of cases may be relieved with Holden's capsules.

Rx: Phenacetin	gr. 2.5	to 5
Aspirin	gr. 2.5	to 5
Atrophine Sulphate	gr. 1/150	to 1/100
Codeine	gr. 1/4	to 1/2

One of these capsules should be given just before the onset of the period followed by two capsules every four hours until the

period of pain has passed. In the mild to moderate cases these capsules may be used without the codeine. In the severe cases the codeine may have to be increased. The use of codeine in the capsules, in patients who have to remain at work, has the disagreeable effect of making them somnolent and as a result frequently has to be omitted. In a fair number of cases this is all that will be necessary, if found insufficient, heat in the form of Elliott's treatment for thirty minutes daily for one week before the onset of menstruation has proved efficacious and should be tried. It should be used before three or four periods for the best results. Heat in the form of hot vaginal douches, electric pads and hot Sitz baths may be of help. Many authors recommend viburnum, given over a long period of time, three times daily for two months in hot water. This is supposed to cause a relaxation of the spastic uterine muscle.

It has been observed that some severe cases of dysmenorrhea which do not respond to ordinary therapy have been relieved by marital life or pregnancy.

Reynolds and Novak believe that an "abnormal amount of theelin with a diminished amount of progestin causes dysmenorrhea" (theoretical). This increased quantity of theelin is supposed to heighten the irritability of the uterine musculature and they believe this heightened irritability is the immediate factor in the production of pain. They recommend the prolan B-like hormone in dysmenorrhea. This luteinization hormone from the urine of pregnant women, is supposed to luteinize the epithelium of the ripe follicles and thus produce the necessary progestin. If the new commercial progestin proves of value clinically it should be tried in these cases.

Surgery may be required for the severe cases that have not been relieved by these procedures. A dilatation and curettage with the insertion of a stem pessary for three periods should be the first procedure.

Warning. Do not use a pessary that is longer than ⅔ the length of the uterus. Sew it in place with nonabsorbable suture material. If a longer pessary is used the continuous contractions of the uterus will force the pessary out too soon.

If the pessary fails, resection of the presacral nerves has proved effectual even in the most stubborn cases. It is not very difficult technically and the results are frequently brilliant.

The induction of a temporary amenorrhea with *x*-rays as a treatment for dysmenorrhea has proved valuable not only as a therapeutic agent but also for its diagnostic value. If the pain does not stop with the amenorrhea one must search elsewhere than the uterus for the etiology of the pain.

Menopausal Symptoms

The menopausal symptoms are due to endocrinological changes. In most cases the symptoms of hot flushes, sweating, irritability, etc., are self-limited and frequently yield to some mild form of therapy. The use of theelin and thyroid seems to help these patients. One may experiment with the various products and combination of products until the right one for the particular patient is found. With the glandular therapy one should also employ diet regulation, bowel hygiene and some form of nerve sedative, as the barbitals and bromides. The psychic factor in the control of their symptoms must constantly be borne in mind.

Question: What relationship has the thyroid to menstrual disorders?

Answer: All patients presenting themselves with menstrual disorders should have a basal metabolism test performed and if found below normal, thyroxin grain $\frac{1}{320}$ to $\frac{1}{160}$ twice daily should be given depending upon the rate. If the metabolic rate is increased and the patient is suffering from hyperthyroidism, mild or severe, the menstrual disorder may be disregarded and the thyroid dysfunction treated.

Question: What is the relationship of obesity to menstrual disorders?

Answer: Obesity in a large percentage of cases is due to overeating, and in a much smaller percentage it is due to some glandular dysfunction. All of these patients should be placed on a low carbohydrate, low fat, high protein diet and to overcome the asthenia they should receive calcium lactate grains 20

every morning on an empty stomach, e.g., before breakfast. If the basal metabolic rate is below normal they should receive thyroxin gr. 1/320 to 1/160 twice a day.

Question: Would you give *x*-ray stimulation to all amenorrheas?

Answer: Yes, after an eighteen month period during which all tests and treatments described have been tried.

Question: Are there any glandular products which would "wind up the menstrual clock"?

Answer: Numerous products have been tried without avail. The commercial houses are constantly trying to produce a prolan A-like substance, a follicle stimulating hormone. There are some on the market at the present time but their efficacy is very questionable.

In this connection it is surprising to note the small quantity of ovarian tissue that may be left in a patient at operation and still have her menstruate normally and yet patients with infantile uteri and two normal appearing ovaries do not menstruate. Apparently something more than the ovaries is necessary for regular menstruation. If this other stimulating substance, whether it be prolan A, *x*-ray or what not, could be given the patient, menstruation could be reestablished.

[*To be continued in subsequent issues.*]

AUTHOR INDEX TO VOLUME XXXVI

Armstrong, W. P. Jr., 332
Arons, Isadore, 533
Astley, G. Mason, 362

Babcock, W. Wayne, 3
Ballenger, Edgar G., 174
Bandler, Clarence G., 162
Barber, Robert F., 386
Bassen, Edward J., 93
Bate, John T., 500
Beisler, Simon A., 213
Belson, Maurice O., 221
Bender, Morris S., 462
Best, R. Russell, 611
Rochner, S. Judd, 279
Borsher, Irving P., 398
Bowen, Arthur, 694
Boyers, L. M., 623
Breidenbach, Lester, 308

Cahill, George F., 653
Caldwell, John A., 736
Charache, Herman, 551
Charlton, M. R., 703
Church, Reynold Erskine, 57
Coakley, Walter A., 50
Coombs, James Norman, 366
Cooper, Henry S. F., 11
Cowett, Max P., 262
Curtis, Dean, 122

Dealy, Frank N., 132
DeCourcy, Joseph L., 283
DeOrsay, Ralph H., 313
Desjardins, Arthur U., 603
Dickinson, Arthur M., 663
Dinnerstein, Morris, 421
Donald, D. C., 514
Drummond, Alvin C., 180
Druss, Joseph G., 102
Dunn, Edward P., 44
Dunn, Fred Squier, 83
Dworkin, Martin, 403

Elder, Omar F., 174
Epstein, Ervin, 472

Falk, Henry C., 31, 559, 750
Fardelmann, Adolf Von Prief, 527
Ferguson, L. K., 270, 313
Ferguson, L. Kraeer, 313
Festerling, E. G., 360
Fett, Herbert C., 288
Frazier, W. D., 672

Gilbert, Judson B., 711
Glass, Morris, 487
Glenn, Frank, 734
Gordon, Charles A., 418
Gordon, Donald, 107
Graham, James, 339
Greene, Lloyd B., 204

Haas, S. L., 720
Hamby, Wallace B., 732
Hamilton G. Johnson, 240, 618
Hark, F. W., 69
Harris, Franklin I., 443
Hawkes, Stuart Z., 398
Henderson, John, 125
Hicken, N. Frederick, 611
Hurley, Vincent, 259
Hyams, Joseph A., 189

Iason, Alfred H., 376

Jeck, Howard S., 197

Kaminester, Sanford, 700
Kassebohm, Fred A., 491
Katz, Abraham, 520
Kelly, Joseph D., 77
Key, J. Albert, 466
Kickham, Edward L., 178
Kirschenmann, J. J., 349
Kirshbaum, J. D., 476

Leddy, Eugene T., 603
Levy, Edward, 244
Lichtenstein, Manuel E., 620
Ludlum, Walter D. Jr., 520
Lynch, Jerome Morley, 240, 618

Mabry, Charles B., 292
MacAusland, Andrew R., 320
Magoun, James A. H., 717
Mason, James B., 505
Mathews, Harvey Burleson, 233
Mathieu, Albert, 219
Mayer, Edgar, 403
McCarty, Claude E., 531
McCauley, John C. Jr., 303
McDonald, Harold P., 174
McNealy, R. W., 620
Mecray, Paul M., 270, 313
Meyer, Herbert Willy, 724
Milbert, Arthur H., 162
Morgan, C. Naunton, 252
Morse, Joseph L., 137
Mulholland, John H., 323
Murphy, Charles C., 384
Murphy, Hugh L., 370
Myerson, Mervin C., 151

Narat, Joseph K., 54, 550, 729
Neef, Frederick Emil, 231

O'Connor, Bernard A., 544
Olin, Harry A., 476
O'Sullivan, John R., 544

Phaneuf, Louis E., 226
Pratt, Gerald H., 118
Preston, Robert L., 298

Rapoport, E., 249
Rathbun, N. P., 739
Rosenwasser, Harry, 96
Ryan, Thomas J., 708

Sammis, Frank, 46
Scannell, John M., 26
Schmier, Adolph A., 389
Schnepp, Kenneth H., 351
Schreiber, Milton J., 491
Shands, A. R., 679
Smyth, Calvin M. Jr., 505
Sokoloff, Boris, 533
Solomon, David I., 737
Som, Max L., 462
Soresi, A. L., 668
Spiesman, Manuel G., 17
Steel, William A., 37
Stern, Elias Lincoln, 509
Stevenson, Clyde A., 603
Stout, Gurn, 412
Straith, Claire L., 88

Tamerin, Joseph A., 91
Thomas, H. B., 69
Thorek, Phil, 22
Tilley, Albert R., 421
Toll, Robert M., 328
Tollman, J. P., 611
Torpin, Richard, 172
Turner, Horace E., 385

Updegraff, Howard L., 94, 414

Vickers, Denver M., 73

Wagner, Jerome, 266
Walkling, Adolph A., 705
Weaver, Hiram C., 523
Weeks, Carnes, 421
Weeth, Charles Reed, 159
Weiskittel, Richard J., 145
Welton, Thurston, 14
Wescott, H. H., 290
White, Alfred S., 443
Whitman, Royal, 553
Woodruff, I. Ogden, 421
Wyeth, George A., 63

Zaph, S. D., 476

SUBJECT INDEX TO VOLUME XXXVI

(Bo.B.) = Bookshelf Browsing; (E.) = Editorial; (S.) = Serial

Abnormalities of rectum, congenital, 240
Abortion, treatment of 487
Abscess, ischiorectal, 279
  peritonsillar, 159
Acute lymphangitis, 37
  pyogenic parotiditis, 125
  regional (terminal) ileitis, 708
  retention of urine, 162
Adhesive, use of, in orthopedic practice, 290
Alcohol; chronic painful conditions amenable to relief by intraspinal (subarachnoid) injection of, 509
Ambulatory saphenous ligation, 398
Anastomoses; a new insufficient and technique for intestinal, 544
Anastomosis, new instrument for intestinal, 734
Angina pectoris and congestive failure, total thyroidectomy in, 421
Angiomata of sketetal muscle, 69
Ano, fissure in, 244
Appendix, mucocele of, with pseudomucinous degeneration, 523
Application of radium in carcinoma of uterine cervix, 231
Artificial pneumothorax, 403
Auditory canal, foreign bodies in external, 96

Bartholin's gland abscess or cyst; office treatment of, 219
Bedsores (decubitus), 17
Benign tumors of back, 145
Bicornuate uterus, pregnancy in rudimentary horn of, 700
Bilary tracts, morbidity and mortality following operations on, 505
Biopsy of uterine cervix, 226
Bites, human, 44
Bladder and urethra, rupture of, 653
  removal of foreign bodies from urethra and, 197
Blank cartridge cannon shot wound of leg, 724
Blood transfusion, 26
Boils and carbuncles, treatment of, 107
Bone fixation, device for tightening wire for, 736
Book Reviews:
  Aural therapy in relation to deafness (Fraser-Harris), 745
  Diseases of the coronary arteries and cardiac pain (Levy), 747
  Recent advances in laryngology and otology (Stevenson), 745
  Recent advances in genitourinary surgery (Bailey), 416
  Textbook of general surgery (Cole), 745
  Textbook of surgical nursing (Keller), 747
  Textbook of surgery (Homans), 746
  The eye and its diseases (Berens, Ed.), 746
  The thyroid: surgery; syndromes: treatment (Sloan), 416
  Urology (Keyes & Ferguson), 416
Bookshelf Browsing:
  Sketch of the evolution of American orthopedic surgery, 553
  The life and times of Thomas Sydenham, 731
Breast; mammographic recognition of intracystic papilloma of, 611
Bunion, treatment of hallux valgus, 332
Burns, 50
  treatment with brilliant green, 54

Cancer of ureter, primary. Studies of the natural history of genitourinary tumors, 711
Carbuncles and boils, treatment of, 107
Carcinoma of uterine cervix; application of radium in, 231
Caruncle, urethral, 178
Cecostomy, simple technique for, 620
Cervix, application of radium in carcinoma of uterine, 231
  biopsy of uterine, 226
Chronic painful conditions amenable to relief by intraspinal (subarachnoid) injection of alcohol, 509
Cinders and splinters, 94
Circumcision; dorsal slit; constriction of male meatus; phymosis and paraphymosis, 174
  simplified anatomical technique, 172
Claw foot and hammer-toe, 351
Clinical comparison of sclerosing solutions in injection treatment of varicose veins, 389
Clips, Michel, in skin coaptation, 14
Clubfoot, treatment of congenital talipes equinovarus, 339
Coccyx, fractures of, and coccygodynia, 303
Cod-liver oil in skin ulcerations; local application of, 472
Colles's fracture, 320
Combined roentgenotherapy and ultra-short wave, 533
Concealment of scars, 91
Congenital abnormalities of rectum, 240
Congestive failure and angina pectoris, total thyroidectomy in, 421
Constriction of male meatus; dorsal slit; phymosis and paraphymosis; circumcision, 174
Cryptitis, 249

Dangers of roentgenoscopy and methods of protection (E.), 603
Device for tightening wire for bone fixation, 736
Diagnosis and treatment of stricture of male urethra, 189
Distention; use of pitressin for control and relief of, 672
Dorsal slit; constriction of male meatus; phymosis and paraphymosis; circumcision, 174

Editorials:
  Dangers of roentgenoscopy and methods of protection, 603
  Maternal mortality and obstetric conscience, 418
Embolism, pulmonary, 694
Endocervicitis; pathology and office treatment of chronic, 233
Epigastric hernia causing severe symptoms, 703

Facial scars, 88
Failure, congestive, and angina pectoris; total thyroidectomy in, 421
Feeding formula; useful artificial, 412
Felon (Whitlow), 366
Femur and hip; first aid treatment in fracture of, 323
Fibroids (S.), 559

Fingers; plastic restoration for loss of all, of both hands, 720
First aid treatment of fracture of femur and hip, 323
Fish hook embedded in finger; removal of, 384
Fissure-in-ano, 244
Fistula, gastrojejunocolic, 527
salivary, 122
Foreign bodies in external auditory canal, 96
rectum, 266
tracheobronchial tree, 462
removal of, from urethra and bladder, 197
Fracture, Colles's, 320
of femur and hip; first aid treatment of, 323
Fractures, malunited, of lower end of humerus, 679
of coccyx and coccygodynia, 303
mandible and maxilla, 83
neck of femur, insertion of Smith-Peterson nail for intracapsular, 466
nose, 77
ribs, 308
some aids in treatment of compound, 551
use of unpadded plaster in treatment of, 292
Frost-bite: treatment by passive vascular exercise, 370
Furuncle of upper lip, 118

Ganglion; pathology and treatment of, 313
Gas gangrene, 22
Gastrojejunocolic fistula, 527
Gastrostomy, inframural, 668
Graft: new method of transfer of full-thickness skin; Grille, 46

Hallux valgus (bunion), treatment of, 332
Hammer-toe and claw foot, 351
Hand infections: office treatment, 376
Head, infections of, 57
malignant tumors of, 63
Heart, right ventricle; stab wound of, 520
Heater for intravenous solutions, 550
Helpful drug in treatment of tuberculosis of urinary bladder, 500
Hemorrhage following tonsillectomy, 151
Hemorrhoids, 252
injection treatment of internal, 259
Hernia, epigastric, causing severe symptoms, 703
the truss in relationship to diagnosis and injection treatment of inguinal, 443
Hip and femur; first aid treatment in fracture of, 323
Hirschpring's disease and polyposis; sympathectomy for, 531
Human bites, 44
Humerus, malunited fractures of lower end of, 679
Hydrocele and varicocele: operative and injection treatment, 204
Hydronephrosis due to ball-valve obstruction from bullet lying free in renal pelvis, 717
Hymen, imperforate, 221
Hyperemesis gravidarum, 491

Ileitis, acute regional (terminal), 708
Imperforate hymen, 221
Incisions of drum membrane in otitis media, 102
Infections of head, 57
neck, 73
Inframural gastrostomy, 668
Infusions, intravenous, 31
Ingrown toe-nail, 349
Injection and operative treatment of hydrocele and variocele, 204
treatment of inguinal hernia, the truss in relationship to diagnosis and, 443
internal hemorrhoids, 259
Injection treatment of varicose veins; clinical comparison of sclerosing solutions, 389
Injuries of the knee, office surgical treatment of, 328
Injury, traumatic, of abdomen with extensive damage to colon and small bowel, 514
Insertion of Smith-Petersen nail for intracapsular fractures of neck of femur, 466
Intestinal anatomoses; a new instrument and technique for, 544
prolapse through enterostomy following Rammstedt operation, 729
Intravenous infusions, 31
solutions, heater for, 550
Ischiorectal abscess, 279

Keloids, warts and moles, 137
Knee, office surgical treatment of injuries of the, 328
Kut-Klamp: a new surgical instrument which simultaneously cuts and retains suture, 737

Leg, blank cartridge cannon shot wound of, 724
Life and times of Thomas Sydenham (Bo.B.), 739
Lip, furuncle of upper, 118
Local application of cod-liver oil in skin ulcerations, 472
Lymphangitis, acute, 37
Lymphosarcoma of stomach: clinical and roentgenological aspects, 476
Lynch's simplification of perineal excision of rectum, 618

Malignant tumors of head, 63
Malunited fractures of lower end of humerus, 679
Mammographic recognition of intracystic papilloma of breast, 611
Mandible and maxilla, fractures of, 83
Maternal mortality and obstetric conscience (E.), 418
Mechanical holder for retractors in neurosurgery, 732
Meckel's diverticulum and volvulus of right colon, 705
Michel clips in skin coaptation, 14
Minor and office surgery (E.), 1
surgery of spine, 298
Moles, warts and keloids, 137
Morbidity and mortality following operations on biliary tract, 505
Mucocele of appendix with pseudomucinous degeneration, 523
Muscle; angiomata of skeletal, 69
Muscles and tendons, rupture of, 283

Neck, infections of, 73
Neurosurgery; mechanical holder for retractors in, 732
New instrument and technique for intestinal anastomoses, 544
for intestinal anastomosis, 734
method for transfer of full-thickness skin: Grille graft, 46
Nose, fractures of, 77

Obstetric conscience and maternal mortality (E.), 418
Orthopedic surgery; sketch of the evolution of American (Bo.B.), 553
Otitis media, incisions of drum membrane in, 102
Office treatment of Bartholin's gland abscess or cyst 219
injuries of the knee, 328
Operating room signals, 414

Papilloma of breast; mammagraphic recognition of intracystic, 611
Paraphymosis and phymosis; circumcision; dorsal slit; constriction of male meatus, 174

Paronychia, 362
Parotiditis, acute pyogenic, 125
Passive vascular exercise; frost-bite: treatment by, 370
Pathology and office treatment of chronic endocervicitis, 233
ganglion, 313
Peptic ulcers, subacute perforations of, 663
Peritonsillar abscess, 159
Phymosis and paraphymosis; circumcision; dorsal slit; constriction of male meatus, 174
Pilonidal cysts: excision and primary suture in ambulatory patients, 270
Pitressin, use of, for control and relief of distention, 672
Plaster-of-Paris, 288
use of unpadded, in treatment of fractures, 292
Plastic restoration for loss of all fingers of both hands, 720
Pneumothorax, artificial, 403
Polydactylism, 385
Polyposis and Hirschsprung's disease; sympathectomy for, 531
Practical clinical gynecology (S.), 559, 750
Pregnancy in rudimentary horn of bicornuate uterus, 700
Pruritus ani, 262
Pulmonary embolism, 694
Puncture wounds, simple treatment for, 360

Radium, application of, in carcinoma of uterine cervix, 231
Rectum, congenital abnormalities of, 240
foreign bodies in, 266
Rectum; Lynch's simplification of perineal resection of, 618
Removal of fishhook embedded in finger, 384
foreign bodies from urethra and bladder, 197
Retention of urine, acute, 162
Retractors, mechanical holder for, in neurosurgery, 732
Ribs, fractures of, 308
Roentgenoscopy, dangers of, and methods of protection (E.), 603
Roentgenotherapy and ultra-short wave, combined, 533
Rupture at bladder and urethra, 653
muscles and tendons, 283

Salivary fistula, 122
Saphenous ligation, ambulatory, 398
Scars, concealment of, 91
facial, 88
Sclerosing solutions in injection treatment of varicose veins; clinical comparison of, 389
Shock, 623
Shot wound of leg, blank cartridge cannon, 724
Signals, operating room, 414
Simple technique for cecostomy, 620
treatment for puncture wounds, 360
Skeletal muscle, angiomata of, 69
Sketch of the evolution of American orthopedic surgery, (Bo.B.), 553
Skin, full thickness, new method for transfer of; Grille graft, 46
Smith-Petersen nail, insertion of, for intracapsular fractures of neck of femur, 466
Some aids in treatment of compound fractures, 551
Spine, minor surgery of, 298
Splinters and cinders, 94
Stab wound of heart, right ventricle, 520
Stomach, lymphosarcoma of: clinical and roentgenological aspects, 476
Stricture of male urethra; diagnosis and treatment, 189
Studies of the natural history of genitourinary tumors. 1: Primary cancer of ureter, 711
Styes, 93
Subacute perforations of peptic ulcers, 663
Sydenham, the life and fines of Thomas, (Bo.B.), 739
Sympathectomy for Hirschsprung's disease and polyposis, 531

Talipes equinovarus (clubfoot), treatment of congenital, 339
Tendons and muscles, rupture of, 283
Thyroidectomy, total, in angina pectoris and congestive failure, 421
Toe-nail, ingrown, 349
Tonsillectomy; hemorrhage following, 151
Total thyroidectomy in angina pectoris and congestive failure, 421
Tracheobronchial tree, foreign bodies in, 462
Transfusion, blood, 26
Traumatic injury of abdomen with extensive damage to colon and small bowel, 514
Treatment of abortion, 487
boils and carbuncles, 107
burns with brilliant green, 54
congenital talipes equinovarus (clubfoot), 339
hallux valgus (bunion), 332
open infected wounds, 11
specific urethritis—new modality, 180
varicose veins and ulcers, 386
Truss in relationship to diagnosis and injection treatment of inguinal hernia, 443
Tuberculosis of urinary bladder; helpful drug in treatment of, 500
Tumors of back, benign, 145
head malignant, 63

Ulcerations; local application of codliver oil in skin, 472
Ulcers and varicose veins, treatment of, 386
subacute perforations of peptic, 663
Ultra-short wave and roentgenotherapy, combined, 533
Ureter, primary cancer of. Studies of the natural history of genitourinary tumors, 711
Urethra and bladder; removal of foreign bodies from, 197
rupture of, 653
diagnosis and treatment of stricture of male, 189
Urethral caruncle, 178
Urethritis, treatment of specific—new modality, 180
Urinary bladder, helpful drug in treatment of tuberculosis of, 500
Urine acute retention of, 162
Useful artificial feeding formula, 412
Use of adhesive in orthopedic practice, 290
pitressin for control and relief of distention, 672
unpadded plaster in treatment of fractures, 292
Uterine cervix; application of radium in carcinoma of, 231
biopsy of, 226

Varicocele and hydrocele; operative and injection treatment, 204
Varicose veins and ulcers; treatment of, 386
clinical comparison of sclerosing solutions in injection treatment of, 389
Vas deferens: operative treatment, 213
Volvulus of right colon and Meckel's diverticulum, 705

Warts, moles and keloids, 137
Wens, 132
Wounds and their complications, 3
treatment of open infected, 11